ACRP Advisory Committee on Radiological Protection

CCRP Comité consultatif de la radioprotection

Advisory Committees of the Atomic Energy Control Board of Canada — Secretariat: Ottawa, Canada K1P 5S9

Comités consultatifs de la Commission de contrôle de l'énergie atomique du Canada — Secrétariat: Ottawa, Canada K1P 5S9

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A REVIEW OF RADIATION RISK ESTIMATES

by the

Advisory Committee on
Radiological Protection

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REVIEW OF RADIATION RISK ESTIMATES

Abstract

Three authoritative reports (UNSCEAR-1988, BEIR-V-1990, and ICRP-1990 Recommendations) on risk estimates have been reviewed and compared to previous risk estimates published by the same organizations. The ICRP now uses the term "probability" in place of the term "risk". For fatal cancers, the new ICRP probability estimates are 5.0×10^{-2} Sv^{-1} for a population of all ages and 4.0×10^{-2} Sv^{-1} for a population of working age. For serious hereditary effects summarized over all generations, the ICRP probability coefficients are 1.0×10^{-2} Sv^{-1} for a population of all ages and 0.6×10^{-2} Sv^{-1} for a population of working age. For prenatal irradiation, at 8 - 15 weeks after conception, there may be a decrease of 30 I.Q. points per Sv and a risk of cancer which may lie in the range of 2 to 10×10^{-2} Sv^{-1}.

Based mainly on the new probability estimates the ICRP recommends a limit on effective dose of 20 mSv per year, averaged over 5 years (100 mSv in 5 years) with the further provision that the effective dose should not exceed 50 mSv in any single year. For public exposure the ICRP recommends an annual limit on effective dose of 1 mSv. However, in special circumstances, a higher value of effective dose could be allowed in a single year provided that the average over 5 five years does not exceed 1 mSv per year.

Once pregnancy has been declared, the conceptus should be protected by applying a supplementary equivalent dose limit to the surface of the woman's abdomen of 2 mSv for the remainder of the pregnancy and by limiting intakes of radionuclides to about 1/20 of the annual limit on intake.

A brief survey of epidemiological studies of workers and the risks from radon and thoron progeny is also included.

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I. REVIEW OF RADIATION RISK ESTIMATES

A. INTRODUCTION

Largely because of the revised dosimetry of those exposed to the atomic bombing at Hiroshima and Nagasaki and because of a longer follow-up of the Japanese survivors, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1988), the Committee on the Biological Effects of Ionizing Radiation, U.S. National Academy of Sciences (BEIR V), and the International Commission on Radiological Protection (ICRP, 1990 draft), revised their previous risk estimates of exposure to ionizing radiation. The Advisory Committee on Radiological Protection (ACRP) has established a Working Group to summarize the results of the above internationally recognized bodies as well as of other relevant studies. This report forms the basis of advice on current risk estimates by ACRP to the Atomic Energy Control Board.

The risks of radiation discussed in this report deal only with effects that are called stochastic which means that the effects are random or statistical in nature. There is no known threshold of radiation dose for causation of stochastic effects and the probability of developing these effects is linearly proportional to dose. Stochastic effects occurring in an irradiated individual include leukemia, fatal or non-fatal cancer. Irradiation of germ cells which may cause stochastic effects to be transmitted to subsequent generations are known as hereditary or genetic effects. These latter effects comprise a spectrum of disorders in the descendants of irradiated individuals.

Deterministic effects or non-stochastic effects have a threshold radiation dose for their production in irradiated persons and the severity of effects are greater with increasing dose. Some examples of deterministic effects are skin burns, cataracts and clinical indications leading to early death. Deterministic effects are not included in the discussion of radiation risks in this report.

All of the risk estimates discussed in this report refer to potential effects and are derived using prudent assumptions for the purpose of radiation protection; there is no proof that low levels of radiation have any deleterious effects on the health of human populations.

Radiation at high doses (see Table 1 in Annex C) is definitely carcinogenic but no cancers unique to radiation are induced. Leukemia, except for chronic lymphatic leukemia, is one of the prominent radiation-induced cancers followed by malignant tumours of the breast, thyroid, lung and certain other sites. Chronic lymphatic leukemia, squamous cell carcinoma of the cervix and Hodgkin's disease do not appear to be induced by radiation. The minimum latency period for leukemia is about 2 years and is about 5-10 years for other tumours.

The probability that a person exposed to radiation will develop cancer depends on several factors. Host factors, pertinent to the individual, are genetic background, age, and sex. Other factors relate to the conditions of irradiation, the magnitude of dose, the time period over which the dose was delivered and the quality of radiation. Other environmental factors, such as lifestyle and exposure to other toxic agents, can interact with radiation and influence this probability.

Since there is an absence of detailed knowledge of radiation carcinogenesis, one must rely on epidemiological studies of humans exposed to radiation to estimate the probability of an individual developing cancer. To provide risks of radiation-induced cancers in a variety of organs the epidemiological study of about 90,000 atomic bomb survivors in Japan is the major source of information. The atomic bomb survivors are almost equally split into males and females and cover a full range of ages and doses. Useful supplementary information is provided by men in the U.K. treated for ankylosing spondylitis, women treated for cervical cancer with X- or gamma-radiation and women with tuberculosis exposed to X-rays to the breast in the course of fluoroscopic examinations of the chest.

The Japanese survivors in the Life Span Study (LSS) have been followed for 40 years but at least another 40 years will be needed to determine the total risk to this population. However, the information on leukemia is probably now almost complete. Since nearly two-thirds of the LSS group are still alive, it is necessary to estimate projections of lifetime risk of fatalities resulting from radiation-induced cancers. To project the overall cancer risk for an exposed population, models are used that extrapolate over time the data based on only a limited period of the lives of individuals. Two projection models are commonly used, the additive (absolute) model and the multiplicative (relative) model. The BEIR V and ICRP 1990 studies (see Annexes D and J) used modifications of these two basic models. The relative risk model is considered more likely to provide a better estimate of the lifetime projection of cancer risks.

B. SUMMARY OF ANNEXES

1. Previous Estimates of Radiation Risks 1977-87

The estimate of lifetime risk of radiation-induced fatal cancers derived by UNSCEAR in 1977 was 1×10^{-2} Sv^{-1}, by ICRP in 1977 was 1.3×10^{-2} Sv^{-1}, and by BEIR III in 1980 was from $0.7\text{-}2.3 \times 10^{-2}$ Sv^{-1} with probable limits from less than 0.1 to 5.0×10^{-2} Sv^{-1}.

The 1977 UNSCEAR report reviewed the epidemiological and dosimetric data that were available at that time for various groups of persons who had been exposed to high radiation doses. These groups included the Hiroshima-Nagasaki bomb survivors, persons exposed to medical X-rays for the treatment of various diseases, radium dial painters, patients injected with thorotrast, uranium miners and others.

The 1977 ICRP report did not give a detailed review of the epidemiological data; risks were apparently based on an ICRP review of data provided in the 1977 UNSCEAR report. In 1978 the ICRP stated that their estimates were intended to be a realistic assessment of effects of exposure to radiation levels below 50 mSv per year to the whole body.

The 1980 BEIR III report provided a detailed review of the same type of epidemiological data that were considered in the 1977 UNSCEAR report, but placed more emphasis on uncertainties in the theoretical models which could be used for interpretation of the data. The BEIR III report considered quadratic, linear-quadratic and linear dose-response relationships as well as additive and multiplicative models for induction of fatal cancers by low-LET radiation. The majority of BEIR III Committee expressed a preference for the linear-quadratic model, which was essentially the same approach as that used by ICRP in 1977-78.

2. New Dose Estimates for Japanese A-bomb Survivors

Previous risk estimates were based on doses derived by a dosimetry system called the Tentative 1965 Dose (T65D). However, some of the test systems used in developing T65D were inappropriate and not representative of the conditions in Hiroshima and Nagasaki. Reviews of the T65D system resulted in a new dosimetry system called Dosimetry System 1986 (DS86).

The bomb yield at Nagasaki was confirmed to be 21 ± 2 kilotons in the DS86 system, while in the T65D it had been taken as 22 kilotons. However, the estimated yield at Hiroshima was increased to 15 ± 3 kilotons in the DS86 system compared to the 12.5 kilotons in the T65D system.

The large amount of metal in the gun-type assembly in the Hiroshima device resulted in significant softening of the neutron energy spectrum. The effect was to reduce the number of neutrons having sufficient energy (> 1 MeV) to reach the survivors and thus reduced their estimated dose from neutron exposure. The T65D neutron source term for Hiroshima was obtained from measurements using a bare ^{235}U reactor that had a neutron energy spectrum much "harder" than the Hiroshima device. This error in the neutron source term in the T65D resulted in a significant overestimation of neutron dose to Hiroshima bomb survivors. The T65D neutron source term for Nagasaki, using later A-bomb test data, is still believed to be reasonable.

The most significant differences for free-in-air kerma (see Glossary) between DS86 and T65D are for Hiroshima. The DS86 values give a larger gamma-ray kerma due in part to the larger estimated yield, and a much smaller (approximately one-tenth) neutron kerma because of the changed neutron source term and the attenuation of neutrons by humidity (underestimated in the T65D). In Nagasaki, the DS86 neutron kerma is somewhat smaller than for T65D while the gamma kermas are in fairly close agreement. The large reduction in the neutron component of radiation at Hiroshima in the DS86 system means that the bomb survivor data can no longer be used to estimate the relative biological effectiveness of neutrons for human carcinogenesis.

The average shielding transmission factors (see Glossary) for gamma rays in Japanese houses are substantially reduced using DS86, resulting in a corresponding average decrease in estimated gamma-ray doses to survivors. Changes in transmission factors for neutrons are considerably smaller and in the opposite direction.

The transmission factors for gamma rays through body tissues in order to calculate organ dose are higher in the DS86 than in the T65D system for gamma-rays, neutron and neutron-capture gammas. Thus the change in the shielding transmission factor which is lower for the DS86 gamma rays, and the change in organ dose transmission factor which is higher for the DS86 gamma rays, tend to cancel each other out.

The risk estimates derived using DS86 are based primarily on exposure to gamma radiation. The large reduction in the estimated neutron component of dose in Hiroshima using DS86 has resulted in the risk estimates for Hiroshima now being much closer to those for Nagasaki. The new (DS86) doses for survivors at Hiroshima and Nagasaki are similar to the T65D doses when expressed as absorbed dose in Gy, but are on average about 2 times lower when expressed as effective dose in Sv with a relative biological effectiveness of 20 for neutrons.

3. UNSCEAR 1988 Cancer Risk Estimates

In the Life-Span Study group of atomic bomb survivors, there was a statistically significant increase, with increasing dose, in the frequency of deaths from leukemia, multiple myeloma, cancers of the oesophagus, stomach, colon, lung, breast, ovary, and urinary bladder. The risk for cancers of the oesophagus and urinary tract was only marginally increased. No significant increase as yet is observed for cancers of the gall bladder, pancreas, uterus and prostate or for malignant lymphomas. The study was extended to include other sites of cancer, such as those of bone, pharynx, nose and larynx and skin, but none had a significant dose response relationship.

The results for 9 types of cancers have been assessed from data on Japanese bomb survivors with some confidence. Also the total risk from all types of cancers has been estimated using a simple linear dose-response model for radiation at high dose rate. Two sets of numbers were determined; one was derived from projections based on the additive (absolute) risk model and the other from projections based on the multiplicative (relative) risk model. The total cancer mortality risk coefficient for the average individual (also averaged over both sexes) is 4.5×10^{-2} Gy^{-1} on the absolute risk model and 7.1×10^{-2} Gy^{-1} on the relative risk model. Lifetime risk estimates based on age specific risk coefficients for whole populations range from 4×10^{-2} Gy^{-1} (absolute model) to 11×10^{-2} Gy^{-1} for the relative risk model. For the working population (aged 25-64 years) the absolute risk model yielded a risk estimate of 4×10^{-2} Gy^{-1} and the multiplicative risk model gave a value of 8×10^{-2} Gy^{-1}.

The Committee agreed that there was a need for a reduction factor to assess these risks for low doses and low dose rates. They believed this dose rate effectiveness factor (see Annex E) to be between 2 and 10.

4. BEIR V 1990 Cancer Risk Estimates

The major part of the BEIR V report is concerned with cancer risks caused by exposure to low-LET radiation (see Glossary). The Committee carried out fairly extensive statistical analyses of the original data from a number of epidemiological studies of humans exposed to ionizing radiation in order to arrive at risk estimate projections for the U.S. population. Statistical modelling techniques were used to analyze these data which allowed for modifying the effects upon risk of time-related factors, such as age at exposure, and time since exposure.

Separate analyses were conducted by the BEIR V Committee for a number of specific cancer sites. To estimate lifetime excess cancer mortality risks the Committee developed preferred models separately for leukemia, respiratory tract, breast, digestive organs and all other cancers combined. In general, the linear relative risk model proved to be the preferred model, with modification by sex, and time dependent factors which varied with the particular site being considered. Only for leukemia was the preferred dose-response a linear-quadratic model. The constant additive risk model did not provide a good fit for any of the cancer sites analyzed and the Committee concluded that this model was no longer tenable.

The modified relative risk model included a term which allowed for the decrement of the probability of a fatal cancer with time after exposure. Different parameters were used for different cancer types so that the form of decrement could be varied to fit the data available. The analysis then provided for age specific coefficients in 10 year intervals for each of the sites selected for study. The results show a very substantial variation in predicted risk with age for most types of cancer, a steady decline with age for excess cancers in digestive organs and breast, but an increase in middle age (reaching a maximum at age 45-55) for respiratory cancer. Women were moderately more radiosensitive than men.

The Japanese survivors of the atomic bombing of Hiroshima and Nagasaki (LSS) are the major source of human data. However, a number of difficulties in estimating leukemia risks from data on Japanese bomb survivors have been noted.

The total risk (averaged for men and women) for all cancers for a dose of 0.1 Sv is given as 0.8×10^{-2}. The contribution for leukemia has been reduced by a factor of 2, from the value expected at 1 Sv at high dose rates but no dose rate effectiveness factor was applied for other fatal cancers.

5. The Effect of Dose Rate on Risk Estimates for Low-LET Radiation

The NCRP report 64 provides a comprehensive summary of the literature on dose rate effectiveness factors (DREFs)[1] related to hereditary and carcinogenic effects. The report concludes that a DREF of between 2 and 10 could be inferred on the basis of radiobiological data when the absorbed dose is 0.2 Gy or less, or the dose rate is 0.05 Gy per year or less. Caution should be exercised when applying a DREF to human risk estimates. Although there is ample evidence for such an effect in cellular and animal systems, there is little supporting human data.

The BEIR V report, in using a linear quadratic model for leukemia, implicitly applied a DREF of 2.1 to the leukemia risk estimate. For all other cancers a linear relationship was used with no DREF. Although the BEIR V report provided estimates of DREF values, it did not recommend any particular value for induction of cancers other than leukemia by low-LET radiation. The summary of the report provided a risk estimate for death from cancer following an acute dose of 0.1 Gy of low-LET radiation and indicated that "for low-LET radiation, accumulation of the same dose over weeks or months, however, is expected to reduce the lifetime risk appreciably, possibly by a factor of 2 or more."

The 1988 UNSCEAR report stated that a DREF could be in the range of 2 to 10 when extrapolating carcinogenic effects from high doses and high dose rates of low-LET radiation to low dose rates.

The 1990 ICRP report applied a single value of 2 for the dose and dose rate effectiveness factor (DDREF)[2] in extrapolating risk coefficients obtained at high doses and high dose rates to ones estimated at low doses and low dose rates.

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[1] DREF: a factor used to derive risk estimates of exposure to low-LET radiation at low dose rates, from risk estimates calculated from data associated with exposures at high dose rates (see Table 1, Annex C).

[2] DDREF: a factor used to derive risk estimates of low doses of low-LET radiation at low dose rates, from risk estimates calculated from data associated with large doses at high dose rates.

6. Prenatal Estimates

It has long been recognized that the mammalian embryo and foetus has a much higher degree of radiosensitivity than the young or adult of the species.

Effects of prenatal irradiation may be classified as "short-term", "long-term" and "late". Short-term effects are those which appear early at the cellular or tissue level or both. When the initial damage is sufficiently great, it may result in malformation or spontaneous abortion (miscarriage). Long-term effects are often biologically-modified expressions of previously induced defects. They include reduced organ and body growth, impaired cell maturation, as well as structural and functional defects, especially of the central nervous system. Radiation-induced disorders of the central nervous system such as severe mental retardation are the result of radiation effects on the proliferation, migration and synaptogenesis of neurons in the brain. The potential late effects of intrauterine irradiation include carcinogenesis and hereditary damage.

Much of the evidence about prenatal radiosensitivity comes from extensive studies of different animal species (primarily rodents) experimentally irradiated in utero under controlled conditions. There are considerable difficulties in directly extrapolating in utero animal radiosensitivity data to humans. Regardless of different organ and tissue transition times, however, the sequence of qualitatively similar differential steps is identical in mammals and humans.

The incidence of severe congenital abnormalities in human populations is about 6% of live births and this estimate is increased to 8.5% when less severe anomalies are included. Environmental factors, including ionizing radiation, may account for about 6% of this total incidence (i.e., for about 0.5% of all live births).

a) Prenatal and Neonatal Deaths

Mortality depends upon the period during gestation at which exposure occurs. It is suggested that 1 Gy, 1.5 Gy and 3 Gy may cause mortality following exposure at 1 week, 5-7 weeks and 21 weeks to term. On the basis of animal studies, there is evidence to suggest a threshold of 0.1 Gy or less for lethal effects to the embryo.

b) Malformations

During the period of organogenesis, malformations may be caused in organs under development at the time of exposure. These effects are deterministic in character with a threshold of about 0.05 - 0.1 Gy, on the basis of animal studies.

c) Severe Mental Retardation

The greatest increase in incidence of severe mental retardation may occur when the foetus is exposed between 8 and 15 weeks, which is the most radiosensitive period for this effect in humans. The risk of severe mental retardation because of exposure during this period is about 40×10^{-2} Gy^{-1} of low-LET radiation with a threshold of 0.4 Gy. The risk during the less sensitive period of 16-25 weeks is 10×10^{-2} Gy^{-1}. This risk is based on the Japanese population exposed to high dose rates. No dose rate effectiveness factor (DREF) has been applied since there is absence of comparative data between high and low dose rate effects.

d) Reduced Intelligence Quotient scores

For individuals exposed during 8-15 weeks after fertilization, and to a lesser extent those exposed during 16-25 weeks, the mean test scores suggest a progressive linear shift downwards in I.Q. scores with increasing exposure. Those exposed to 1 Gy during the most sensitive period of brain development between 8 and 15 weeks exhibit a loss in intelligence as measured by I.Q. testing of about 30 points. A similar but smaller shift is detectable in those irradiated in the period from 16 to 25 weeks. No evidence of a radiation-related effect on intelligence among those individuals exposed within 0-7 weeks after fertilization or in the 26th or subsequent weeks was obtained.

The shift in I.Q. appears to follow a linear non-threshold dose-effect relationship. This conclusion is tenuous because the available data are very limited. All the observations on I.Q. and severe mental retardation relate to high dose and high dose rate and their direct use probably overestimates the risk at low dose and low dose rates.

e) Childhood Cancers and Leukemias

Irradiated foetuses seem to be susceptible to childhood leukemias and other childhood cancers which are usually expressed before the age of 10. There is disagreement between the data from the Japanese bombing survivors prenatally exposed to large doses, who do not show an excess of childhood cancers, compared with the data from large populations of children who received small doses of prenatal radiation for various medical reasons and who show an excess of childhood cancers. The estimated risk of childhood cancers and leukemias up to the age of 10 years ranges from 2×10^{-2} Sv^{-1} to 10×10^{-2} Sv^{-1} or possibly even higher.

7. Genetic Risk Estimates

There is substantial evidence that radiation-induced genetic changes in lower organisms are truly stochastic, but no reliable evidence for the induction of excess genetic disorders in the children of humans exposed to high radiation doses exists. There is no statistically significant increase in genetic disorders among the children born to parents of whom at least one was a Hiroshima-Nagasaki survivor, although there are some trends in the data which suggest that the normal rate of genetic mutations might be doubled by a radiation dose of 2 Gy.

Scientific committees responsible for quantitative assessment of the genetic risks of radiation have relied on other methods of estimating these risks. The basic requirements include a measure of the normal incidence of genetic disorders in human populations, the contribution of new mutations in each generation to these incidences, and the probable rate of induction of new mutations per unit dose of radiation.

The 1977 ICRP recommendations estimated that the total probability of radiation-induced genetic disorders in the children and grandchildren of exposed persons was 1×10^{-2} Sv^{-1} received prior to conception. Since the mean age of parents at reproduction is about 30 years, the genetic risk averaged over the complete population with a life expectancy of 70 years would be 0.4×10^{-2} Sv^{-1}.

The 1990 ICRP recommendations suggest that radiation-induced genetic diseases should be summed over all subsequent generations rather than over the first two generations only. Using some reasonable assumptions, the probability of all genetic diseases summed over all subsequent generations would be about 1 x 10^{-2} Sv^{-1} averaged over a complete population with a life expectancy of 70 years. Since the working population is exposed to occupational sources of radiation only from age 18 on, the total genetic risk should be somewhat smaller, about 0.6 x 10^{-2} Sv^{-1}.

A recent study by Gardner has suggested a possible association between occupational exposure to fathers and development of childhood leukemia in their children. This appears to be at variance with the Hiroshima-Nagasaki data noted above, but if confirmed, this association might require an appreciable increase in genetic risk estimates. The ACRP will keep this topic under review.

8. Epidemiological Studies of Workers

Estimates of the probability of developing cancer after exposure to low doses of radiation at low dose rates are based on data derived from Hiroshima and Nagasaki survivors who were exposed to high doses at high dose rates. There are many uncertainties involved in this extrapolation. These uncertainties would disappear if it were possible to derive risk estimates directly from the study of workers exposed to low doses of low-LET radiation at low dose rates.

Epidemiological studies of radiation workers usually show lower rates of cancer mortality than observed for persons of the same age and sex in the general population. In general, the differences are not statistically significant and the observable decreases are attributed to healthy worker effects.

The BEIR V report concluded that the studies of radiation workers "have provided no evidence to date that risk estimates for leukemias and other types of cancer combined are in error, based on extrapolation from high dose studies". Gilbert and co-workers indicated "that estimates obtained through extrapolation from high doses data do not seriously underestimate risks of low-dose exposure, but leave open the possibility that extrapolation may overestimate the risk". Nevertheless the data on radiation workers are compatible with the DREF of a magnitude which falls in the ranges suggested by UNSCEAR and BEIR V.

Low levels of radiation exposure are not a major factor affecting cancer mortality; other factors appear to play a more important role. It is generally considered reasonable and prudent to assume a linear non-threshold dose-response relationship for the probability of radiation-induced cancer; this assumption is unproven at low doses and low dose rates and is, in fact, unprovable for very small increments in dose. For this reason, it would be prudent to speak of potential health detriments of exposure to low levels of radiation.

9. Risks of Radon and Thoron Progeny

Studies of workers in a wide variety of uranium and other mines in different countries have consistently shown excess lung cancers which have been attributed to high concentrations of radon progeny in these mines. There are at present 7 studies from which quantitative estimates of excess lung cancers per unit exposure to radon progeny can be derived. These risk estimates cover a seven to ten-fold range.

The lung cancer data from the epidemiological studies of miners are compatible with a linear non-threshold dose-response relationship at least up to a cumulative exposure of 300 WLM. The minimum latent period of lung cancers is 5 years after first exposure. The maximum increase in lung cancers occurs 10-15 years after exposure, after which the incidence of lung cancers decreases to approach zero 20-30 years after exposure. The lung cancers in Hiroshima and Nagasaki exposures appear to continue for at least 40 years after exposure.

An average lifetime risk of 3 x 10^{-4} WLM^{-1} might be considered reasonable based on data for adult male miners. This average risk per WLM is equivalent to that from about 18 mSv of whole body gamma radiation using the weighted risk coefficient recommended by ICRP in 1977, but would be equivalent to that from about 5 mSv using the detriment coefficient given in the 1990 ICRP recommendations. ICRP and UNSCEAR previously used a conversion factor of 10 mSv per WLM.

There are many uncertainties in the extrapolation of these lifetime risk estimates for miners to a whole population of all ages and both sexes. Attempts to do this have resulted in values of 2 x 10^{-4} to 3.5 x 10^{-4} WLM^{-1}. A risk of 3 x 10^{-4} WLM^{-1} for the general population would mean that one WLM would be equivalent to that from about 4 mSv of whole body gamma radiation using the aggregated detriment coefficient for the whole population given in the 1990 ICRP recommendations.

There are no epidemiological data from which the risk from inhalation of thoron progeny can be derived. Dosimetric calculations suggest that the risk per WLM of thoron progeny is about one-third per WLM of radon progeny.

10. Summary of ICRP 1990 Recommendations

a) Total Stochastic Risk

The direct use of epidemiological data obtained at high doses and high dose rates will probably overestimate the extrapolated risks for all radiogenic fatal cancers at low doses and low dose rates. For this reason ICRP has introduced the Dose and Dose Rate Effectiveness Factor (DDREF) to correct this overestimate. ICRP has decided to use a factor of 2 to reduce the probability of observed effects at high doses and high dose rates to give estimates of the probability of effects at low doses and low dose rates. The DDREF has been included in risk coefficients for doses below 0.2 Gy and for higher absorbed doses when the dose rate is less than 0.1 Gy per hour of low-LET radiation.

Another major difficulty in interpreting human data is that of estimating the number of fatal cancers yet to appear in populations being studied when a majority of the population is still alive. For leukemia and bone cancer, there is no major difficulty because the rate of appearance of new cases has fallen almost to the expected rate in the control populations. For other cancers, however, the incidence is still greater than that expected in controls. To account for these differences, ICRP has adopted the multiplicative risk model for cancers other than leukemia. In addition ICRP has used a modified multiplicative model which was used by the U.S. National Institute of Health (NIH) to develop its radioepidemiological tables. The latter takes an absolute risk from the Japanese population, transfers it to another population as an absolute risk and converts it to a relative risk in the other populations. Populations considered are those in Japan, U.S., U.K., Puerto Rico, China, and the average of all five populations.

Data relating to high doses and high dose rates are interpreted as suggesting a lifetime probability coefficient for fatal cancers in a reference population of both sexes and of working age of about 8×10^{-2} Sv^{-1} and the corresponding value for whole populations, including children, of about 10×10^{-2} Sv^{-1}. Using a DDREF of 2, the nominal risk coefficient for the working population is 4×10^{-2} Sv^{-1} and for the whole population is 5×10^{-2} Sv^{-1}. Because of uncertainties in estimating cancer risks from radiation exposure and because the ICRP estimated risks for defined populations with defined exposure patterns, it calls the estimated probability of fatal cancers per unit effective dose the nominal fatal probability coefficient.

The risk coefficient for serious hereditary effects is 0.6×10^{-2} Sv^{-1} for the working population and 1.0×10^{-2} Sv^{-1} for the whole population.

The risk coefficient for mental retardation as a result of in utero irradiation 8 - 15 weeks after conception is 30 I.Q. reduction points per Sv.

Adoption of the relative risk projection model and revision of the dosimetry for the Japanese bomb survivors are the major reasons that the previous cancer risk estimates used by ICRP as the basis for radiation protection appear now to have increased overall by a factor of 3-4 since 1977.

b) Cancer Risk Estimates for Individual Organs

ICRP took the probabilities of fatal cancers in specific organs, as determined by UNSCEAR and BEIR V on the Japanese population, and with two projection models, the multiplicative and the NIH modified multiplicative model, recalculated the probabilities of fatal cancers in individual organs for five populations. The probabilities for individual organs were converted to fractions of total risk and averages of these fractions of total risk were averaged over the five populations. To minimize statistical variations the average results of the two projection models were averaged again. Data for fatal cancers of bone, liver, skin and thyroid were derived not from studies on Japanese bomb survivors but from various other epidemiological studies. The risk coefficients for the whole population are given in Table 1 where they are compared with those derived from ICRP-26.

The normal incidence of stomach cancer is higher in Japan than in the U.S.A., while the reverse is true for breast cancer. Table 1 therefore includes estimates of the probability of radiation-induced cancer as calculated from the values given for the U.S. population only in Appendix B of the 1990 ICRP draft.

c) Derivation of Tissue Weighting Factors (W_T)

In addition to providing risk estimates for fatal cancer, the ICRP attempted to quantify the risk due to non-fatal cancer. To account for different radiosensitivities of tissues and organs on the probability of developing stochastic effects, the ICRP has given numerical values for tissue weighting factors (see Table 2 in Annex J). The tissue weighting factors (W_T) include three components:

(1) the probability and time of occurrence of attributable fatal cancers;
(2) the probability and time of occurrence and relative severity of attributable non-fatal cancers; and
(3) the probability of, and loss of normal life expectancy due to, severe hereditary effects summed over all subsequent generations.

The tissue weighting factors do not differ significantly for the whole population and the working population. They relate to a human population of equal numbers of males and females and a wide range of ages. When account is taken of fatal and non-fatal cancers and severe hereditary effects summed over all subsequent generations, the nominal aggregated detriment coefficients are about 7 x 10^{-2} Sv^{-1} for the whole population and 5.6 x 10^{-2} Sv^{-1} for the working population.

Table 1

Probability of fatal cancers (10^{-2} Sv^{-1}) in a population of both sexes and all ages after exposure to low dose, low dose rate low-LET radiation

	ICRP 26 (1977[a])	New probabilities of fatal cancer (1990[b])		
		International average	U.S.A.: multiplicative model	U.S.A.: NIH model
Bone marrow	.20	0.50	0.54	0.40
Bone surface	.05	0.05	(0.05)	(0.05)
Lung	.20	0.85	1.15	0.53
Thyroid	.05	0.08	(0.08)	(0.08)
Breast	.25	0.20	0.42	0.15
Colon		0.85	1.79	0.82
Oesophagus		0.30	0.08	0.11
Stomach		1.10	0.18	1.38
Liver		0.15	(0.15)	(0.15)
Bladder		0.30	0.43	0.21
Ovary		0.10	0.17	0.10
Skin		0.02	(0.02)	(0.02)
Remainder	.50	0.50	0.54	0.35
Total	1.25	5.0	5.6	4.35

a IC77 ICRP Publication 26. Recommendations of the International Commission on Radiological Protection. Annals of the ICRP, Vol. 2, No. 3 (1977).

b IC91 1990 Recommendations of the International Commission on Radiological Protection. International Commission on Radiological Protection (1991).

d) Dose Limits

The 1990 ICRP draft has recommended an occupational dose limit of 100 mSv per 5 years, which is equivalent to an average of 20 mSv per year to workers. The average loss of life expectancy due to fatal cancers that might be induced by continued exposure to 20 mSv per year for 47 years is calculated on the basis of the relative (multiplicative) risk projection model to be similar to the corresponding values for the 1977 limit of 50 mSv per year using an additive risk projection model, and is similar to that attributable to 47 years of employment in an industry with a fatal accident rate of 3 per 10 000 workers per year.

The recommended limit for members of the public from planned exposure situations (excluding medical treatments) is 1 mSv per year averaged over any consecutive 5 years.

The basis for the control of occupational exposure of women who are not pregnant is the same as that for men. A dose of 1 mSv to the foetus is roughly equivalent to about 2 mSv of external radiation to the abdomen of the mother. To protect the conceptus the ICRP recommends that a supplementary equivalent dose limit to the surface of the woman's abdomen of 2 mSv for the remainder of the pregnancy be applied. Once pregnancy has been declared, the intake of radionuclides should be limited to one-twentieth of the (new) annual limit on intake for non-pregnant workers.

C. DISCUSSION

1. Recent Cancer Risk Estimates

Table 2 summarizes the estimates of risk of radiation-induced fatal cancers as given in recent reports of three scientific committees. These estimates are primarily dependant on about 295 excess fatal cancers in a total of 1119 observed between 1950 and 1985 in Japanese bomb survivors who received more than 0.2 Gy in 1945. There is no statistically significant excess of leukemias or of all other fatal cancers among the bomb survivors who received less than 0.2 Gy. Information on excess cancers in other populations who received high radiation doses in the past has had little impact on the particular estimates given in Table 2.

Since the majority of the Japanese bomb survivors were still alive in 1985, various risk projection models were used to estimate the number of excess fatal cancers that may appear during the remaining lifetime of this population. The 1988 UNSCEAR report used additive and relative risk models for this purpose, while the 1990 BEIR V report used a modified relative risk model in which the presumed effects are dependant on age at exposure and time since exposure. The recent ICRP draft suggested that a relative risk projection model was more appropriate than the additive model and adopted a risk estimate which is 3 to 4 times greater than that recommended by the ICRP in 1977. This increase is due largely to two factors: adoption of a relative risk projection model and revision of the Hiroshima-Nagasaki dosimetry. The ICRP projections suggest a lifetime total of 800-900 excess fatal cancers among the 11 000 to 12 000 Japanese bomb survivors who received 0.2 Gy or more in 1945.

2. Uncertainties in Cancer Risk Estimates

There are uncertainties in the choice of the risk projection models used to estimate lifetime risk of radiation-induced fatal cancer from observations on excess cancers in a population of whom the majority of persons are still alive. When the effects of age at exposure, sex, and time since exposure are taken into account, the relative and absolute risk projection models fit the data on excess cancer mortality among the Japanese bomb survivors equally well. If one chooses to use only various relative or modified relative risk projection models, the projected risks as calculated by the three committees listed in Table 2 all fall in the region of 6-11 x 10^{-2} Gy^{-1} for the whole population when exposed to low-LET radiation at high dose rate. The average value of 10 x 10^{-2} Gy^{-1} selected in the 1990 ICRP draft is conservative but compatible with this range.

Other uncertainties appear to be associated with leukemia risk estimates; these uncertainties are associated mainly with the fitting of observed data to an assumed dose-response relationship for radiation-induced leukemia. The 1988 UNSCEAR report used the best linear non-threshold fit to all available data points for the Japanese bomb survivors, while the 1990 BEIR V report fitted data points below 4 Sv to a linear-quadratic dose-response relationship. Other differences involve methods for estimating "missing" data on excess leukemias in the period 1947-49 and expression of doses in terms of absorbed dose in Gy or effective dose in Sv. The net result is a leukemia risk estimate which is roughly 2 times higher at 1 Gy (Sv) or roughly 5 times higher at 4 Gy (Sv) in the BEIR V report than in the 1988 UNSCEAR report for radiation exposures at high dose rate. There appear to be unresolved problems in the derivation of the leukemia risk estimates given in the BEIR V report. The 1990 ICRP draft, which had the benefit of both the BEIR V and UNSCEAR reports, preferred to use the 1988 UNSCEAR approach.

Table 2
Estimates of excess fatal cancers induced by exposure to low-LET radiation at high and low dose rates

	UNSCEAR 1988	BEIR V 1990	ICRP 1990
Projected risk at 1 Gy at high dose rate (x 10^{-2}):			
whole population:	4 - 11	9	10
workers:	4 - 8	7	8
Projected risk per Sv at low dose rate, assuming a DREF of two (x 10^{-2}):			
whole population:	2 - 5.5	4.4	5
workers:	2 - 4	3.5	4

A major uncertainty is associated with extrapolation from effects observed at high dose rate to those predicted at low dose rate with low-LET radiation. All three of the committees noted in Table 2 agree that the carcinogenic effects of low-LET radiation are likely to be lower at low dose rate than at high dose rate. The 1988 UNSCEAR report suggested a dose-rate effectiveness factor (DREF) of 2 to 10. The BEIR V report discussed a DREF of 4-5 for cancers other than leukemia but did not recommend any particular value. The 1990 ICRP draft adopted a DDREF of 2. This choice represents a rather conservative value in view of the data discussed in Annex E.

The choice of DREF would be simplified if it were possible to measure a reproducible increase in cancer mortality in workers exposed to low-LET radiation at low doses and low dose rates. No statistically significant increases of this kind have been observed to date. Two of the three major studies available to 1989 on workers exposed to low-LET radiation suggest, rather, a decrease in cancer mortality with increasing accumulated exposure. Caution is needed in the application of ICRP cancer risk estimates to actual populations of radiation workers. It would be prudent to speak of potential health detriments of exposure to low levels of low-LET radiation.

The nominal probability estimates proposed in the 1990 ICRP draft indicate that the probability of death from cancer could potentially be increased from the normal value of 25% for all Canadians to about 29% in workers exposed in theory to 20 mSv per year for 47 years. To date, the follow-up of radiation workers exposed to low-LET radiation provides no clear evidence of the suggested increase.

3. Weighting Factors

The term radiation weighting factor (W_R) is used in the 1990 ICRP draft to replace the term quality factor. There is little change in the proposed values. Alpha particles and fast neutrons in the range of 0.1 to 2 MeV are assigned radiation weighting factors of 20. Previous ambiguities were clarified by the recommendation that X-rays, gamma rays and electrons of all energies be assigned a weighting factor of 1.

It should also be emphasized that the nominal probability coefficients proposed in the 1990 ICRP draft are intended to be averages for populations in countries with an average life expectancy of 70-80 years. The probability that an individual person might develop cancer as a result of exposure to radiation depends on a variety of factors. Host factors pertinent to the individual include genetic background, age, and sex. Other environmental factors such as life style can also influence the probability. These factors were considered briefly in the 1982, 1986 and 1988 UNSCEAR reports and are the topic of research in various laboratories, including those at Chalk River in Canada. It is also known that the probability of radiation-induced lung cancer is not necessarily influenced by age and by cigarette smoking habits in the same manner for external gamma radiation as for inhaled radon progeny. Caution is thus essential in applying these nominal probability coefficients to individual workers; for purposes of workers' compensation, this topic has been considered in ACRP-5 ("Assessment for medico-legal purposes of the contribution of occupational or other defined exposure to ionizing radiation as causative agent in individuals suffering from or having died of cancer").

Tissue weighting factors (W_T) have been changed appreciably from those recommended by the ICRP in 1977. This draft proposes weighting factors for 12 individual organs or tissues, as compared to 6 in 1977. There is considerable difference in the weighting factors for certain organs, notably for the stomach, depending upon the risk projection model used in transferring data from the Japanese population to other populations such as those in the USA (Table 1). The proposed ICRP tissue weighting factors are intended to represent an international average. The nominal risk for irradiation of bone marrow was doubled to allow for the fact that the average time between exposure and death is shorter for radiation-induced leukemia than for most other radiation-induced fatal cancers. Some allowance was made for non-fatal or curable cancers that might be induced by radiation. The nominal risk for irradiation of the gonads was approximately doubled from 0.4×10^{-2} Sv^{-1} in 1977 to 0.6×10^{-2} Sv^{-1} for workers or 1×10^{-2} Sv^{-1} for the whole population in the 1990 draft, to allow for serious radiation-induced genetic diseases in all subsequent generations rather than in the first two generations only. The net result is that the fatal cancer coefficient for workers is increased from 4×10^{-2} Sv^{-1} (Table 2) to an aggregated detriment of 5.6×10^{-2} Sv^{-1}, while the aggregated detriment for the whole population becomes 7.3×10^{-2} Sv^{-1} in the 1990 ICRP recommendations. The fatal cancer coefficient represents about 70% of the aggregated detriment in both cases.

4. New Dose Limits and Implications

As a result of the increase in nominal risk estimates for fatal cancers and the accompanying increase in weighted effect coefficients, the 1990 ICRP draft has recommended an average dose limit of 20 mSv per year for radiation workers and of 1 mSv per year for planned exposures (excluding medical treatments) of members of the general public. The average loss of life expectancy attributable to a radiation-induced fatal cancer in workers is calculated to be about 13 years on the basis of the new relative risk projection model, as compared to about 20 years for an additive risk projection model. Thus the average loss of life expectancy for a hypothetical worker exposed to 20 mSv each year from age 18 to age 65 is calculated on the basis of the 1990 data to be similar to that predicted for an exposure to 50 mSv per year for 47 years on the basis of the 1977 data using an absolute risk model.

The new dose limits, if endorsed in Canada, will necessitate recalculation of annual limits on intake and derived air concentrations for many radionuclides. New annual limits on intake are provided in ICRP Publication 61. Because of proposed changes in tissue weighting factors (as well as dose limits), annual limits on intake for workers would not necessarily be reduced by a simple factor of 2.5 since some radionuclides are not uniformly dispersed in all tissues. In certain cases, current limits on intake are based on a non-stochastic or deterministic limit of 0.5 Sv per year to a given tissue since this was more restrictive than a limit based on presumed stochastic effects; because the deterministic limit is no longer limiting, there may be little change in the annual limit on intake for these particular radionuclides, despite the change in annual dose limit based on nominal stochastic risks. Changes in annual limits on intake would of course also impact on the "scheduled quantities" as designated by the AECB for a variety of radionuclides.

Calculations of annual limits on intake of those alpha-emitting radionuclides which concentrate in bone are likely to be conservative. The nominal risk estimates for 3 tissues, as given in the 1990 ICRP draft, are compared with those given for three alpha-emitters in the 1988 BEIR IV report (Table 3). The risk estimates for bone surfaces and liver are in reasonable agreement, which is to be expected since the 1990 ICRP draft derived their values for these two tissues from data on populations exposed to alpha emitters, not from data on Japanese bomb survivors. However, the leukemia risk estimates derived from data on the Japanese bomb survivors are much higher than those which have been derived from data on populations exposed to thorotrast, radium-224 or radium-226 (Table 3).

There are major problems in calculating annual limits on intake of alpha emitters concentrated in bone. These problems involve the slow accumulation of long-lived radionuclides in bone over periods of several decades, the burial of long-lived alpha emitters deep in bone due to bone remodelling, and the possibility that low concentrations of long-lived alpha emitters such as radium-226 (Table 3) in the bone may not produce any excess cancers within the normal life span of humans. Any calculated values for the annual limit on intake of long-lived alpha emitters which are based primarily on concentrations in bone should therefore be viewed with extreme caution.

Dose limits for inhaled radon progeny will require special attention. The 1990 ICRP draft says little on this topic but does suggest a nominal lifetime risk coefficient of 1-4 x 10^{-4} WLM^{-1}. If one assumed that the average risk coefficient were 2.5 x 10^{-4} WLM^{-1}, then an average whole body dose of 20 mSv per year would be equivalent to about 4.5 WLM inhaled radon progeny per year. This topic has been considered in more detail in ACRP-12 ("Radiological hazards to uranium miners") and will presumably receive further attention from the ICRP in the near future.

Another consequence of the change in nominal stochastic risks for external radiation may be a recalculation of the effective dose equivalent or "effective dose" received by the general population from radon. The 1988 UNSCEAR report attributes 1.4 mSv per year to inhaled radon progeny from natural sources while the 1990 BEIR V report attributes 2 mSv per year to this source. Both of these numbers may be decreased appreciably, possibly by a factor of about 2, as a result of the suggested increase in risk estimates for gamma and beta radiation from natural sources. This decrease will come about as a result of a change in conversion factor from 10 mSv per WLM to about 5 mSv. This topic will require further attention.

Table 3

Fatal cancer risk estimates (x 10^{-4} Sv^{-1}) for 3 tissues in the general population as derived from the 1990 ICRP draft and from observations on populations exposed to alpha-emitters (1988 BEIR IV report), assuming a radiation weighting factor of 20 for alpha particles

	ICRP 1990	BEIR IV		
		Thorotrast	Ra-224	Ra-226
Bone surface	5	2.8 - 6	10	Possible threshold
Bone marrow (leukemia)	50	2.5 - 3	No clear evidence of excess	No significant excess
Liver	15	13 - 15	No estimate	No estimate

An important change in the 1990 ICRP draft recommendations is the restriction on occupational exposures of pregnant women. The February 1990 draft indicated that "once pregnancy has been declared, the dose to the conceptus should be subject to a dose limit of 5 mSv during the remainder of pregnancy, with no more than about 1 mSv in the period from 8 to 15 weeks after conception". The primary purpose of the latter part of this recommendation was to ensure that the average I.Q. of the exposed child would not be decreased by more than 0.03 I.Q. points from the average value of 100, and that the normal incidence of severe mental retardation would not be increased appreciably. In the November 1990 ICRP draft, the ICRP recommended that to protect the conceptus a supplementary equivalent dose limit to the surface of the woman's abdomen of 2 mSv be applied for the remainder of the pregnancy and to limit the intake of radionuclides to about one-twentieth of the annual limit on intake. If implemented, in the opinion of ACRP this recommendation would mean that the pregnant mother should in practice be removed from essentially all work with radiation and radioactive materials because of practical problems in measuring and controlling radiation exposures to the required degree of accuracy.

The dose limit of 1 mSv per year (average) for members of the general public might raise problems as to who should be designated as a radiation worker, particularly in hospitals and nuclear medicine departments. The 1990 ICRP draft has suggested that workplaces should be designated as controlled areas and supervised areas, either at the design stage or locally by the operating management on the basis of operational experience and judgement. Controlled areas require the workers to follow well-established procedures aimed at controlling radiation exposures, while a supervised area is one in which the working conditions are kept under review but special procedures are not normally needed. Individual monitoring for external radiation is fairly simple and should be used for all those who are occupationally exposed, unless it is clear that their doses will be consistently low. Individual monitoring for intakes of radioactive material should be used routinely only for workers who are regularly employed in areas that are designated as controlled areas specifically in relation to the control of contamination. The classification of occupationally exposed workers requires further study.

The 1990 ICRP draft has commented on potential misapplications of the recommended dose limits. These dose limits are not intended to be regarded as a line of demarcation between safe and dangerous, nor are they intended to be seen as the most simple and effective way of keeping exposures low. Moreover, they are not intended to be incorporated as regulatory instruments when the optimization of protection may be a more appropriate course of action. Dose limits are intended as a limiting restriction on each individual's accumulation of dose and are not appropriate to all types of exposure under all circumstances.

The 1990 ICRP draft distinguishes between exposures resulting from planned practices and those resulting from other activities. The new recommendations on dose limits are applicable in the case of planned exposures. The principles of justification and optimization of exposures should be applied in all situations, but the recommended dose limits should not be applied to medical exposures of patients, to exposure from natural sources or to exposures in emergency situations. After due consideration of the ICRP suggestions, it would seem reasonable that the new dose limits of 20 mSv per year (average) to workers and 1 mSv per year to members of the general public should be incorporated immediately into the design of all future nuclear facilities. Some existing facilities may find it difficult to comply with new dose limits; these facilities should be reviewed on a case by case basis with the view of achieving target dose constraints.

The 1990 ICRP draft has further suggested that the term occupational exposures should be extended to all exposures incurred at work as the result of situations that can reasonably be regarded as being under the control of the operating management. This would include operations of commercial jet aircraft and operations in hard rock mines, spas and other workplaces where high concentrations of radon progeny from natural sources are probable. Implementation of this recommendation would require close cooperation between various federal and provincial agencies in Canada.

In the control of occupational exposures directly due to an accident, the aim should be to keep the doses within those permitted in normal conditions, but this may not be possible in serious accidents. The 1990 ICRP draft recommends that exposures of emergency teams during emergency and remedial action should be limited to 500 mSv (or 5 Sv to the skin) except for life-saving actions. The intent of this recommendation is to prevent any permanent non-stochastic or deterministic damage to body tissues of emergency workers. It might be noted that the threshold for death due to acute radiation exposures of healthy workers is in the region of 2 Sv (UNSCEAR 1988). Although derived intervention levels for use in a serious nuclear emergency should be promulgated by, or on behalf of, the regulatory agency, the 1990 ICRP draft recommends that these intervention levels should not be treated as limits.

The 1990 ICRP draft has limited its recommendations on radiation exposures of members of the general public primarily to planned exposure situations and to general advice on exposures during medical treatment of patients as well as general advice on intervention levels in the event of unusually high exposures from natural sources or in the event of a serious nuclear accident. The latter intervention levels provide a useful way of structuring the procedures of radiological protection but should not be treated as dose limits. The basic principles are that any proposed intervention will do more good than harm (justification) and that the form, scale and duration of the intervention should be chosen so as to optimize the protection. It is further suggested that, although by far the largest component of public exposure is due to natural sources, this provides no justification for reducing the attention paid to smaller but more readily controlled exposures to artificial sources. A previous ICRP publication in 1984 (ICRP Publication 39) included exposure to radon progeny from natural sources as a readily controllable exposure.

D. CONCLUSIONS AND RECOMMENDATIONS

The ACRP has reviewed three recent authoritative reports on risk estimates by UNSCEAR (1988), BEIR V (1990) and ICRP (1990 February, June and November drafts). These reports were a result of revised dosimetry of radiation from the A-bombs detonated at Hiroshima and Nagasaki, the longer follow-up of the atomic bomb survivors, the preference for projection of cancer fatalities by the relative risk model, and new data from other epidemiological studies. It should be pointed out that the new risk estimates are largely based on the Japanese atomic bomb survivors, the so-called Life Span Study. The ACRP recommends that the new dose limits based on new risk estimates should be adopted immediately by the AECB.

ACRP has reached the following conclusions:

1. **Risks of Stochastic Effects**

(a) **Fatal Cancers**

The total potential fatal cancer probability for workers has been increased by a factor of about three times over the previous ICRP-26 estimate of 1.25×10^{-2} Sv^{-1}. Using a dose and dose rate effectiveness factor of 2, the ICRP 1990 draft gives nominal probability coefficients of 5.0×10^{-2} Sv^{-1} for a population of all ages and 4.0×10^{-2} Sv^{-1} for a population of working age. These values are generally compatible with those stated by BEIR V (1990) and UNSCEAR (1988) and should be adopted subject to the following caveats. The nominal probability coefficients are applicable for exposure to low-LET radiation delivered at low doses and low dose rates to the defined populations. For more homogeneous subpopulations the use of age-specific probability coefficients, such as given in BEIR V, should be considered. However, there are unresolved problems in the derivation of leukemia probability estimates in the BEIR V document and ACRP recommends those given in the ICRP 1990 draft report be used. The ACRP wishes to draw attention to the many uncertainties in deriving lifetime probability estimates for the potential effects of low doses and low dose rates from the limited data available. However, it would still be reasonable and prudent to apply the new risk coefficients in radiation protection.

(b) **Serious Hereditary Effects**

For serious hereditary effects summed over all generations, the nominal probability coefficients are 1.0×10^{-2} Sv^{-1} for a population of all ages and 0.6×10^{-2} Sv^{-1} for a population of working age. The increase from the 1977 ICRP recommendations is due largely to the fact that hereditary effects are now summed over all subsequent generations of progeny, rather than over the first two generations only.

(c) **Prenatal Irradiation Effects**

There may be a decrease of 30 I.Q. points per Sv for prenatal irradiation 8-15 weeks after conception. There may be increased probability of cancer from prenatal irradiation; the best estimate of probability lies in the range of $2\text{-}10 \times 10^{-2}$ Sv^{-1} or possibly even higher.

2. As a result of changes in cancer probability estimates and other considerations, the ICRP 1990 draft has recommended that occupational exposure limits should not exceed an effective dose of 100 mSv over 5 years, with an overriding limit of 50 mSv in any single year. This implies an average annual limit of 20 mSv (effective dose).

The new occupational dose limits are lower than those given in current and proposed regulations in Canada. The ACRP does not anticipate problems achieving the new occupational exposure limits in most segments of the nuclear industry with the exception of underground uranium mining. It is recognized that some pre-existing facilities and activities in Canada may find it difficult to comply with the new occupational dose limits but these operations should be reviewed on a case by case basis with plans to be developed for achieving compliance with the new limits as soon as practicable. The ACRP is definitely not in favour of "grandfathering" facilities or activities and recommends that the AECB immediately adopt the new occupational dose limits.

3. The ICRP now recommends that the dose limit for public exposure should be an average effective dose of 1 mSv per year over any period of 5 consecutive years. In some places, such as in hospitals and in nuclear medicine departments, it may be difficult to limit immediately the exposure of some members of the public to an average of 1 mSv per year. Some hospital workers who are not classified currently as atomic radiation workers may receive doses in the range of 1-5 mSv per year.

The ACRP recommends that the AECB adopt the 1 mSv per year dose limit to the public as soon as practicable. Again pre-existing facilities and activities should be reviewed with the aim of developing plans to achieve compliance as soon as practicable.

4. The ICRP draft of November 1990 no longer bases the protection of the foetus from radiation on the risk of preventing severe mental retardation or reduced I.Q. scores. The philosophy has shifted with the intent to protect the foetus broadly as if it were a member of the public. Once pregnancy has been declared, the conceptus should be protected by applying a supplementary equivalent dose limit to the surface of the woman's abdomen of 2 mSv for the remainder of the pregnancy and to limit intakes of radionuclides to about one-twentieth of the annual limit on intake. In practice this more restrictive dose limit during pregnancy may mean the immediate removal of some pregnant women from work involving radiation, for example, in activities with a few highly specialized workers. This may be a controversial and costly recommendation to implement and ACRP gives notice to AECB that it may be faced with many problems as a result of such an implementation. Some of these problems may be economic or infringe on union agreements or on human rights. The ACRP feels that this issue requires further study and recommends that the AECB explore this problem in its socio-economic impact analysis.

To ensure that the foetus does not receive more than 1 mSv from internally deposited radionuclides in the pregnant woman, the ACRP recommends that AECB increase support for research on the dosimetric aspects of internally deposited radionuclides in the pregnant woman.

5. Because of recommended changes in both the tissue weighting factor and dose limits, annual limits on intake of radionuclides will need to be recalculated. The recalculated values may impact on proposed scheduled quantities of various radionuclides.

6. Valuable advice is given in the 1990 ICRP draft on exposures in emergency situations. Exposures of emergency teams during remedial action should be limited to 0.5 Sv effective dose (or 5 Sv to the skin) except for life-saving actions. The ACRP endorses the guidance provided by the ICRP in this respect.

7. Continued international collaboration on the epidemiological studies of workers exposed to low-LET radiation is strongly recommended. The resulting data are expected to assist in minimizing uncertainties concerning the carcinogenic effects of low doses of radiation at low dose rates.

Since there is no absolute proof that low levels of radiation do cause cancer, the ACRP advises that it would be prudent to speak of potential health detriments of exposure to low levels of radiation.

8. The 1990 ICRP draft has suggested that the term occupational exposures should be extended to include all exposures incurred at work as the result of situations that can reasonably be regarded as being under control of operating management. This would include operation of commercial jet aircraft and work places where high concentrations of radon progeny from natural sources are probable. The ACRP urges that the AECB initiate consultation with appropriate provincial and federal agencies to implement required legislation to control the above mentioned exposures.

9. ACRP understands that new limits on exposure to radon progeny will be published by ICRP in a separate document. As a result of changes, it should be noted that conversion factors stating the equivalence of WLM to effective dose may be decreased.

10. Finally, the conceptual framework of radiation protection, namely, justification, optimization, and dose limitation, proposed by the ICRP is endorsed by the ACRP. This framework deals with practices which add doses and risks to people, and intervention which subtracts doses that would otherwise be received. An example of intervention would be planning evacuation in the event of an accident.

II. ANNEXES

ANNEX A

Previous Estimates of Radiation Risks 1977-1987

(D.K. Myers)

Estimates of the risk of radiation-induced stochastic effects published by the International Commission on Radiological Protection (ICRP) between 1977 and 1986 are summarized in Table 1. These quantitative estimates have an important influence on recommended limits for exposures of radiation workers and of the general population to ionizing radiation.

Table 2 summarizes the estimates of lifetime risk of radiation-induced fatal cancers that were derived by the UNSCEAR, ICRP and BEIR III Committees in 1977-86. Review of the methods used to derive these risk estimates may be useful for an understanding of current scientific discussions concerning revision of these estimates.

Table 1

Previous ICRP estimates of probability of radiation-induced stochastic or potentially-stochastic effects (IC77, IC85, IC86)

Biological effect	Risk per 100 person-Sv to exposed persons	Risk per 100 person-Sv averaged over a whole population with an average life expectancy of 75 years
Fatal cancers	1.3 (a)	1.3 (a)
Curable cancers	1.3 - 2.3	1.3 - 2.3
Genetic disorders in children & grandchildren	1.0	0.4
Severe mental retardation after irradiation in utero	10.0	about 0.13

(a) The nominal value of 1.25×10^{-2} Sv^{-1} was rounded up to avoid false impressions of great accuracy.

Table 2

Estimates of probability of radiation-induced fatal cancers in previous reports of scientific committees

Report	Risk per 100 person-Sv
UNSCEAR 1977 [UN77]	about 1
ICRP 1977 [IC77]	1.3
BEIR III 1980 (low-LET radiations only) [BE80]	0.7 - 2.3 (range from less than 0.1 to 5.0)

Annex G in the 1977 UNSCEAR report (UN77) reviewed the epidemiological and dosimetric data that were available at that time for various groups of persons who had in the past been exposed to high radiation doses. These groups included the Hiroshima-Nagasaki bomb survivors, person exposed to medical X-rays for the treatment of various diseases, radium dial painters, thorotrast patients, uranium miners and others. Two major conclusions were derived: (a) At moderately high doses of low-LET radiation above 1 Gy, the risk of radiation-induced fatal cancers appeared to be about 0.5 x 10^{-2} per Gy for leukemia plus 2 x 10^{-2} for other fatal cancers, for a total risk of 2.5 x 10^{-2} per Gy of low-LET radiation (para. 317 in Annex G of UNSCEAR 1977). (b) At lower doses of low-LET radiation, the lifetime risk of radiation-induced leukemia was estimated to be about 0.15-0.25 x 10^{-2} per Gy of low-LET radiation. Evidence was cited to indicate that increased incidence of leukemia had ceased by 30 years after exposure; thus, the observed risk was essentially identical to the lifetime risk of radiation-induced leukemia. Assuming that the lifetime risk of other fatal cancers might be 4-6 times that of leukemia on the basis of earlier mortality studies of American radiologists and of patients exposed to high doses of X-rays for medical purposes (para. 247 in Annex G of UNSCEAR 1977), the total risk for all fatal induced malignancies was estimated at about 1 x 10^{-2} per Sv of low-LET radiation at lower doses of radiation (Table 2).

The 1977 ICRP report (IC77) did not provide a detailed review of the epidemiological data; risk estimates were apparently based on ICRP review of the data provided in UNSCEAR 1977 (UN77). The ICRP report suggested a leukemia risk of 0.2 x 10^{-2} per Sv and a total risk for all fatal induced malignancies of 1 x 10^{-2} per Sv in men or 1.5 x 10^{-2} per Sv in women, the difference being due to the risk of radiation-induced fatal breast cancer in women. This report further indicated that an approximate risk estimate of 10^{-2} fatal cancers per Sv could be used both for radiation workers and for the general public. The initial 1977 document (IC77), like the UNSCEAR report (UN77), was somewhat ambiguous as to whether or not risks at very low dose levels, such as might be encountered from environmental exposures to radiation, might be substantially less. This was, however, clarified in a 1978 ICRP statement (IC78) to the effect that these estimates were intended to be realistic assessments of risks of exposure to radiation levels below 50 mSv per year to the whole body. Thus, the ICRP consolidated its previous suggestions that risks of fatal cancer should be considered to be directly proportional to accumulated radiation dose at low dose rates.

NCRP Report No. 64 (NC80a) provided further theoretical support for the ICRP interpretation of the epidemiological data. The NCRP review of experimental data from other living organisms emphasized the value of the linear-quadratic model for interpretation of dose-response curves on biological effects of exposure to low-LET radiation. At low doses at any dose rate, or at higher doses at low dose rate, effects appear to be directly proportional to accumulated doses of X- or gamma-rays. The effects per unit dose at higher doses delivered at high dose rate are in general 2-10 times greater than effects per unit dose at low dose rate in the case of low-LET radiation (NC80a).

A similar decrease in effects per unit dose is not observed at lower dose rates in the case of high-LET radiation; comprehensive theories explaining these differences have been developed. It should be noted that NCRP Report No. 64 did, however, express reservations about the value of the linear-quadratic model for interpretation of epidemiological data on radiation-induced breast cancer in women. NCRP Report No. 66 (NC80b) also suggested that a simple linear dose-response model provided a good fit to the latter data.

The 1980 BEIR III report (BE80) provided a detailed review of the same type of epidemiological data that were considered in the 1977 UNSCEAR report (UN77), but placed more emphasis on uncertainties in the theoretical models which could be used for interpretation of these data. This BEIR report was also notable for its inclusion of dissenting notes and comments by various members of the Committee.

The BEIR III report (BE80) continued the use of both absolute and relative risk (BE72) models to interpret epidemiological data. The first model reports data in terms of excess number of cancers observed; the second model reports data in terms of percent increase in normal incidence of cancer. These concepts were applied in the 1972 BEIR I report (BE72); the evidence available for review in BEIR I suggested that neither model was necessarily better than the other and that the true risks might lie somewhere in between the predictions suggested by these two theoretical models. If complete lifetime follow-up of exposed groups of persons were available, a choice between absolute and relative risk models would be irrelevant. The question relating to different models for prediction of lifetime risks arises because a large proportion of the exposed cohorts are usually still alive at the time of observation; this is also true for persons who were exposed at younger ages at Hiroshima and Nagasaki in 1945. The majority of the BEIR III Committee members were again unable to make a clear choice in favour of the relative or the absolute risk projection model, and consequently gave the lifetime risk estimates that were calculated by both methods.

The difference in projected lifetime cancer risks using these two models is largely due to a difference in estimated number of radiation-induced cancers which might appear in old age, when the natural incidence of cancers is highest, after exposure at a young age. Differences in average loss of life expectancy are, thus, smaller than the differences in estimated number of radiation-induced cancers (IC85, My80).

The BEIR III (BE80) Committee also considered quadratic, linear-quadratic and linear dose-response models for induction of fatal cancers of low-LET radiation. (The dose-response relationship for high-LET radiation was assumed to be linear at low doses.) In themselves, the epidemiological data were not precise enough to permit a clear-cut choice between these three dose-response models (BE80). The majority of the BEIR III Committee expressed a preference in favour of the linear-quadratic model, which is essentially the same approach that was used by the ICRP in 1977-78.

In the case of radiogenic breast cancer, the BEIR III Committee provided estimates based on a linear dose-response model but noted that the true risks at low doses "could be as low as one-third of the risks corresponding to the linear absolute-risk model, and could conceivably be zero at 1 rad [10 mGY] if the models used ... are not applicable at very low doses". The 1980 BEIR III report obviously left unresolved many practical problems in the estimation of lifetime risk of radiation-induced cancers.

An important addition to epidemiological data on the Hiroshima-Nagasaki survivors resulted from the detectable excess of breast cancers which appeared by 1980 in Japanese women who had been exposed to radiation from atomic bombs in 1945 when they were less than 10 years of age (To84). This excess, which is clearly related to radiation dose, was not seen in earlier surveys and appeared only after the women in question had reached ages at which breast cancer rates normally increase to appreciable levels (To84). This finding has provided a major impetus to adoption of the relative risk model for projection of lifetime cancer risk estimates.

An important revision resulted from a 1981 report indicating major errors in the previous estimates of doses received by the survivors of the Hiroshima-Nagasaki bombs (Lo81). A number of authors attempted to derive preliminary estimates of cancer risks from the Hiroshima-Nagasaki data following the initial publication of these revised kerma doses in 1981. These attempts were generally regarded as premature. The 1982 UNSCEAR report summarized the situation as follows:

> "While it is impossible yet to say exactly what influence the revisions, if accepted, will have on the risk estimates, it is unlikely that this influence will exceed a factor of 2. Indeed, improved agreement between data from Hiroshima and Nagasaki may tend ultimately to strengthen confidence in the estimates. Secondly, the information derived from the survivors of the atomic bombs in the two cities is only one of the sources of human exposure that the Committee has used in arriving at its estimates. While little change is, therefore, expected to result in regard to estimates for cancer induction in man by X- and gamma-rays, an important presumed source of information for whole body neutron irradiation will no longer be available..."

The results of the major reassessment of doses (designated DS86) that has been carried out by groups in Japan, the US, and the UK were published by the Radiation Effects Research Foundation in 1987 (RE87) and these doses are the basis for the dosimetry-related revisions to the risk estimates.

A preliminary estimate of lifetime cancer risks for the Hiroshima-Nagasaki survivors based on the new dosimetry was provided by Preston and Pierce (Pr87). The preliminary estimates suggested a lifetime risk of radiation-induced fatal cancers for a general population of all ages which is much greater than the lifetime cancer risk of 1.3 per 100 Sv suggested in the 1977 ICRP recommendations (Tables 1 and 2) for low radiation doses.

The risk projections of Preston and Pierce and other data (Sh87) were considered at the September 1987 meeting of the ICRP in Como. The ICRP statement from this meeting (IC87) indicated that these new data might "raise the risk estimate for the exposed population by a total factor in the order of 2". The 1987 ICRP statement further indicated that "the risk data are yet far from conclusive", and that the Commission would await the results of ongoing comprehensive evaluation of epidemiological data by UNSCEAR, the BEIR V Committee and the ICRP before judging the consequences for revision of the ICRP system of dose limitation.

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ANNEX B

New Dose Estimates for Japanese A-Bomb Survivors
(D.A. Agnew)

1. HISTORICAL BACKGROUND

1.1 The Period 1945 to Mid 1950s

The bomb called "Little Boy" that exploded 570 meters above the surface of Hiroshima on August 6, 1945 was the only one of its kind ever exploded by the United States. The fissionable material was ^{235}U and it was detonated by uniting 2 subcritical masses in a very large metal gun-type assembly. Three days later a second nuclear device, called "Fat Man", exploded 503 meters above Nagasaki. The fissionable material in this device, ^{239}Pu, was assembled into a critical mass using a high explosive implosion method. After the war, several bombs of the latter type were detonated at the Nevada Test Site, providing important information that was later to be used in assessing the radiation exposure of the Japanese bomb survivors.

Immediately after the bombings, Japanese scientists conducted a large scale study of all aspects of the bombing (SC53). Several of the investigations have provided valuable data for recent dose reassessments. These include the measurement of neutron-induced ^{32}P in sulfur on hydro poles, measurement of radioactive fallout and determination of the hypocentres(1) of the bombs. In addition survivors were identified, their location and movements at the time of the bombings documented, and follow-up health studies initiated. However, it was the mid-1950s before technology reached the stage at which it was considered possible to determine with any real reliability the radiation doses to the survivors.

1.2 Tentative 1957 Dose

The first dose estimates for survivors were designated Tentative 1957 Doses or T57D values. The values were based on experimental measurements of free-in-air kerma related to distance from ground zero and house shielding taken during atmospheric atomic bomb tests at the Nevada Test Site. Because of large uncertainties in the explosive yield estimates (especially for Hiroshima) and locations of the bursts in Hiroshima and Nagasaki, and doubts about the method of estimating shielding from buildings, T57D was never regarded as a definitive dosimetry. The primary use of the T57D system was to identify people who were believed to have been significantly exposed. No systematic program to assess the exposure of the bomb survivors was ever undertaken using T57D.

1.3 Tentative 1965 Dose

In the period following 1956 a number of experiments were performed during some of the last atmospheric A-bomb tests (Operations Plumbbob and Hardtack) and with a bare ^{235}U reactor (Operation BREN) to correct the shortcomings identified in

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[1] Location on the ground directly under the bomb at the time of detonation.

T57D. In 1965, a revised dosimetry system called Tentative 1965 Dose (T65D) was adopted. It was based primarily on the improved free-in-air kerma combined with the distance curves of Auxier (Au66) that now corrected for ground scatter, and house shielding transmission factors of Cheka et al. (Ch65). An excellent description of the development of T65D is given by Auxier (Au77).

1.4 Dosimetry System 1986

The usefulness of the epidemiological data from Hiroshima and Nagasaki depends on the accuracy of the radiation doses assigned to the survivors, and until 1980 it was thought that the uncertainty in these dose estimates using the Tentative 1965 Dose System was about 30%. However, independent reviews of the T65D system by an NCRP Task Group (Ke81) and scientists at the Lawrence Livermore Laboratory (Lo80) discovered significant problems, and so a 5 year program to reassess the doses to survivors was undertaken by American and Japanese scientists. The study resulted in a new dosimetry system called Dosimetry System 1986 (DS86) that is documented in two Radiation Effects Research Foundation publications (Re87, Re88).

2. COMPARISON OF DS86 AND T65D

2.1 Methods of Dose Estimation

The end product of applying the T65D or DS86 system is an estimate of the dose to each individual A-bomb survivor known to be at a specific location some known distance from the bomb hypocentre. To acquire the dose estimate one must, at least in theory, determine the number of neutrons and gamma rays that are generated at the time of detonation, the effects of penetrating the bomb casement and fireball, the interactions of the radiations as they are transported through the air-over-ground, pass through buildings and penetrate the body to deposit energy in specific organs or tissues. In fact DS86 uses sophisticated computer codes and files of physical data to simulate each of these steps in order to arrive at an estimate of dose to each survivor. T65D on the other hand used empirical formulas developed from experimental data collected during atmospheric A-bomb tests and from other experimental systems. As we shall see below, some of the experimental systems used in developing T65D were inappropriate and not representative of the conditions in Hiroshima and Nagasaki.

2.2 Bomb Yield

The accepted method of measuring yield is to perform a radiochemical analysis of the debris from the explosion, but no such measurements were available from Japan. However, the detonations of several Nagasaki-type devices at the Nevada Test Site have provided high confidence in the yield for the Nagasaki bomb. The values for DS86 and T65D are shown in Table 1.

The yield of the Hiroshima bomb for both DS86 and T65D was determined using a variety of absolute methods, such as sulfur activation and blast damage, and relative methods that involve comparing the distances at which equal thermal and blast effects occurred in Hiroshima and Nagasaki. The DS86 theoretical estimate of the yield, 15 kt, agrees with the value recommended in DS86 that represents a weighted average using a variety of methods including those described above.

2.3 Source Term (Leakage Factor)

The Source Term is a description of the number of neutrons and gamma rays emerging from a bomb per kiloton of yield, and their distributions in energy and direction. An important factor in determining the source term is the nature and position of the materials in the bomb. Generating the source terms for DS86 involved combining Monte Carlo techniques that calculate the transport of neutrons and photons through bomb materials with codes that calculate temperature and hydrodynamic motion of parts of the bomb resulting from nuclear energy release. Extensive testing of these codes has confirmed their reliability.

The large amount of metal in the Hiroshima device resulted in significant softening of the neutron energy spectrum. The effect was to reduce the number of neutrons having sufficient energy (>1 Mev) to reach the survivors and thus to reduce their dose from neutron exposure. The T65D neutron source term for Hiroshima was obtained from measurements using a bare ^{235}U static reactor (Operation BREN) that had a neutron energy spectrum much "harder" than the Hiroshima device. This error in the neutron source term in T65D resulted in a significant overestimation of neutron dose to Hiroshima bomb survivors. A reasonable T65D neutron source term estimate was obtained for Nagasaki using A-bomb test data.

2.4 Free-In-Air (FIA) Kerma Versus Distance

DS86 radiation transport codes were used to calculate the neutron and gamma ray fluence and their energy and angular distributions at those locations occupied by survivors at the time their exposure. The results for DS86, showing FIA kerma in tissue (converted from fluence data) as a function of distance from the hypocentre for Hiroshima and Nagasaki, are given in figures 1 and 2. These curves combine the factors of yield, source term and transportation through air-over-ground to give the kerma in a small mass of tissue at a distance from the hypocentre. No account is taken of the shielding from buildings, terrain or body tissue.

FIA tissue kerma versus distance curves used in T65D were obtained empirically by fitting air-ground interface correction factors as well as fluence data obtained in A-bomb tests and BREN experiments to the form

$$F - G_0(1/R^2)e^{-(R/L)}$$

where F - fluence, R - distance to bomb at detonation, L - attenuation length, and G_0 is the fluence at R - 1 meter.

However, test data for T65D was collected at the Nevada Test Site under conditions of very low humidity, while Japan in August 1945 was very humid. Since neutron transport through air is attenuated by moisture (i.e., L, the distance to reduce the fluence by 1/e, is decreased), the T65D data underestimated the attenuation of neutrons and contributed to an overestimation of dose to the survivors in Hiroshima and Nagasaki by a factor of about 2. The FIA tissue kerma versus distance curves for T65D are shown in figures 1 and 2.

The most significant differences between DS86 and T65D are for Hiroshima. The DS86 values give a larger gamma-ray kerma, due in part to the larger estimated yield, and a much smaller (approximately 1/10) neutron kerma because of the neutron source term and humidity errors described above. In Nagasaki, the DS86 neutron kerma is somewhat smaller than for T65D while the gamma kermas are in fairly close agreement. The large reduction in the neutron component of radiation at Hiroshima in the DS86 system means that the bomb survivor data can no longer be used to estimate the RBE of neutrons for human carcinogenesis.

The FIA kerma for neutrons and gamma-rays can be compared to results of neutron activation and thermoluminescence studies performed on building materials in the two cities. The results for measured gamma-ray kerma using thermoluminescence techniques are shown in figures 3 and 4. The measurements support the DS86 calculations over the T65D values. Measurements of ^{32}P in sulfur used as an adhesive for insulators on electric power poles provide a measure of the high energy (>3Mev) neutron fluence at various ground ranges. A graph showing measured activation versus calculated activation (using DS86 program) for Hiroshima is shown in figure 5. The results suggest good accuracy for the neutron fluence calculations with energies in the sulfur activation range and at short distances from the hypocentre. At larger distances the measured values are too uncertain to support any strong conclusion on agreement.

The fluence of neutrons at thermal or near thermal energies have also been measured. Measurements of ^{152}Eu induced in natural europium in rock form a body of experimental data which show no discrepancies in comparison to DS86 calculated values. However, the activation measurements are sufficiently uncertain, especially at distances greater than 1000 meters, that support for the calculated low-energy fluence is weak.

In contrast, measurements of ^{60}Co taken from steel concrete reinforcing bars, when compared to calculated values using the DS86 program, show significant discrepancies (see figure 6). Beyond 500 meters the calculated values are less than the values determined from Co-60 activation. If the measured values for thermal neutrons were accepted as correct, and the assumption made that the calculated neutron fluences are low by a factor that applies at all neutron energies, then the proportion of neutron kerma in the mixed radiation field beyond 1000 meters (where most of the survivors were located) would change from insignificant to significant. The effect would be to reduce the derived risk estimates, and possibly restore the bomb survivor data as a source of information on the RBE of neutrons for human carcinogenesis. Both measurements and calculations have been closely examined by independent researchers and found to be sound. Strong efforts to resolve the cobalt discrepancy have only served to confirm its existence. Although the consensus of expert opinion is that the calculations are correct, the possibility exists that the neutron kerma for Hiroshima and Nagasaki are wrong.

2.5 House and Terrain Shielding

The FIA tissue kerma values discussed above assume a flat air-ground interface and no buildings. Corrections must be made that take into account the shielding provided to a survivor from being located inside a building, or outside but close to a building or geographical feature that can attenuate some of the incident radiation. DS86 performs radiation transport calculations to provide the number, energy and direction of photons and neutrons at any site inside or outside typical Japanese houses. This information can then be used for subsequent organ dose estimations. The shielding calculations include shielding and scatter from nearby houses and the generation of secondary gamma-rays from neutron interactions with building materials.

In T65D the effect of shielding was accounted for by multiplying the FIA kerma by a Transmission Factor (TF). The TFs were obtained from experiments in which replicas of Japanese houses were irradiated using the BREN reactor and a large ^{60}Co source. However, the higher ratio of neutrons to gamma-rays produced by the BREN reactor, relative to the bombs in Japan, resulted in a larger quantity of secondary gamma-rays(a) being measured inside the houses. Therefore, the gamma-ray Transmission Factor (kerma inside house/kerma without house) was overestimated. Also, T65D did not account for changes of TF with increased distance from the blast due to hardening of the neutron spectrum or to a shift in the angle of elevation of the explosion.

The average shielding provided in DS86, expressed as TFs, is shown in Table 2 together with TFs taken from the T65D program. The average transmission factors for gamma-rays in Japanese houses are substantially reduced under DS86 resulting in a corresponding average decrease in gamma-ray dose to survivors. Changes for neutrons are considerably smaller and in the opposite direction.

2.6 Organ Doses

DS86 was designed to calculate the mean scalar fluence in each of 15 target organs in the body of an individual in a specific orientation with respect to the hypocentre, at a specific location inside a house or outside, and in a standing, kneeling or prone position. Modified MIRD-type phantoms were developed to represent the anatomical features of adults and children in Japan in 1945. The calculations include the fluence of secondary gamma-rays produced by the interaction of neutrons in the body. From the mean scalar fluence one can calculate values for tissue kerma, dose or dose equivalent using an appropriate set of conversion factors. The uncertainties in the estimates are due primarily to the uncertainty in the orientation of the individual.

At the time T65D was introduced little was done in the way of calculating organ doses. However, in the 1970s efforts were made to use the simple shielded kerma values to estimate organ doses within simple cylindrical phantoms exposed to isotropic fields. The results obtained using DS86 indicate that these early calculations (presented as T65DR) overestimated the shielding the body provides to the internal organs. The average organ dose transmission factors for 12 organs are shown in Table 3 for both DS86 and T65DR. The transmission factors are higher in the DS86 than in the T65DR system for gamma-rays, neutrons and neutron-capture gammas. Thus the change in the shielding transmission factor which is lower for the DS86 gamma-rays, and the change in the organ dose transmission factor which is higher for the DS86 gamma-rays, tend to cancel each other out.

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[a] Gamma radiation produced by interaction of neutrons with building materials.

A comparison of DS86 and T65DR mean organ-absorbed doses for those survivors exposed to 0.01 Gy or more is given in Table 4 (Sh89). Except for the female breast, the total organ-absorbed doses calculated using DS86 are slightly higher than those determined using T65D. However, risks estimates are calculated using the mean dose equivalent delivered to each target organ. Using Table 4, the mean organ-absorbed dose equivalents have been calculated assuming RBE values for neutrons of 1, 10 and 20.

From the results shown in Table 5 the following conclusions can be drawn.

(i) The risk estimates derived using DS86 are based primarily on exposure to gamma radiation. The exact RBE of neutrons is no longer a critical factor. As seen in Table 5, the change in RBE for neutrons from 10 to 20 increases the DS86 average organ-dose equivalents by only 5 to 15%. Since the risk estimates are inversely proportional to the dose equivalent received, the risk estimates will decrease by a corresponding amount. (The ICRP currently recommends a radiation weighting factor of 20 for fast neutrons with energies in the region of 0.1 to 2 MeV. This value has been applied directly in some of the calculations of risk estimates for the Japanese bomb survivors. However, the radiation weighting factor for fission neutrons may be reduced to considerably lower values by the time that these neutrons have penetrated the body and reached the internal organs (see ICRP Publication 51)).

(ii) The difference in risk estimates derived using DS86 or T65DR (assuming all non-dosimetric factors remain constant) depends on the assumed value of the RBE for neutrons. For an RBE of 10, the risk estimates derived using DS86 will exceed those of T65DR by about 50%. If the RBE is 20, the DS86 derived risk estimates will be approximately twice the T65DR values.

The large reduction in the neutron component of dose in Hiroshima using DS86 has resulted in the risk estimates for Hiroshima now being much closer to those for Nagasaki.

2.7 Radiation Exposure From Residual Radioactivity

Most of the dose to survivors in Hiroshima and Nagasaki came from direct radiations produced by the exploding weapons. There was, however, residual radioactivity produced by neutron activation of materials near the hypocentres, and radioactive fallout of activation and fission products from the cloud formed by the explosion. DS86 was the first attempt to include estimates of exposure to residual radioactivity for the survivors.

It is possible to produce reasonable estimates of exposure from these sources only for external radiation. Although the internal contribution of long-lived fission products appears small, there is no way to evaluate the exposures to short-lived products. Because the radiation measurements made immediately after the detonations were fragmentary, external radiation doses for all individuals exposed to residual radioactivity cannot be developed from the available data. Also, information on the exact movements of all survivors in the areas of residual radioactivity is incomplete. However, the number of individuals in the two cities exposed to residual radioactivity was small, and the individual exposures received are not significantly large compared with the exposures from direct radiation at the time of the bomb.

2.8 Uncertainty Associated With DS86 Doses

The dose estimates made for the Japanese A-bomb survivors are all subject to at least 3 sources of error, namely those that stem from (i) determinations of dose in air with distance from the epicentre, (ii) the attenuation factors for building materials and tissues, and (iii) the location and position of each survivor. For an individual, the magnitude of the uncertainty in the dose estimate depends primarily on the information available in the RERF Data Base For Individual Survivors. For about one half of the proximal survivors (i.e., those within 1600 meters of the hypocentre in Hiroshima and 2000 meters in Nagasaki) the most complete information is available. Their shielding is described using the so-called 9-parameter model. For others, including individuals at greater distances from the hypocentre (distal survivors), specific shielding information is generally not available and so average transmission factors for Japanese houses are used. For those located outside houses at detonation, and who were shielded by buildings or terrain, a less sophisticated method called the Globe Method is used to determine the shielded kerma. The accuracy of the methods of dose estimation can be rank ordered as

(i) 9 parameter method
(ii) average transmission factor for Japanese houses
(iii) Globe method

A list of the factors contributing to the uncertainty of DS86 dose values calculated using the 9-parameter method is given in Table 6. The associated uncertainty values were extracted from reference KA89 and are applicable to the total dose and correspond to one standard deviation of the mean as a percentage of the mean, which is referred to as the fractional standard deviation (FSD). These values are representative of those which apply to a survivor located 1,500 meters from the hypocentre; some factors vary with distance.

The first 3 factors in Table 6 are associated with the calculation of Free-In-Air Kerma. They combine to give an overall uncertainty in the FIA Kerma of 14.7%. A large component of the uncertainty relates to the location of the survivor at the time of the blast and for survivors at 1500 meters is determined to be 21%. It should be noted that the uncertainty associated with location and position of the survivor can never be evaluated rigorously for all of the individuals in the DS86 cohort. The uncertainty in determining the shielded kerma is given as 16.7%. However, as indicated by the values in parentheses, the uncertainty is subject to wide variations depending on the shielding model used. Thus, the total uncertainty in the assigned dose can vary from approximately 29% to 45%.

Table 1

Bomb Yield in Kilotons of TNT

	Hiroshima	Nagasaki
DS86	15 ± 3	21 ± 2
T65D	12.5	22

from reference RE87

Table 2

Average House Transmission Factors by Dose System and City

City	Dose system	Gamma	Neutron
Hiroshima	DS86	0.46	0.36
	T65DR	0.90	0.31
	DS86/T65DR	0.51	1.16
Nagasaki	DS86	0.48	0.41
	T65DR	0.81	0.35
	DS86/T65DR	0.59	1.17

from reference Sh89

Table 3

Average Organ Dose Transmission Factors by Dose System

Organ gamma	Dose system	Gamma	Neutron	Neutron-capture
Bone marrow	DS86[a]	0.81	0.37	0.42
	T65DR[b]	0.56	0.28	0.07
	DS86/T65DR	1.45	1.32	6.00
Colon	DS86	0.74	0.19	0.41
	T65DR	0.40	0.14	0.08
	DS86/T65DR	1.85	1.36	5.13
Stomach	DS86	0.75	0.28	0.40
	T65DR	0.47	0.18	0.07
	DS86/T65DR	1.60	1.56	5.71
Lung	DS86	0.80	0.33	0.37
	T65DR	0.50	0.22	0.07
	DS86/T65DR	1.60	1.50	5.29
Female Breast	DS86	0.85	0.61	0.32
	T65DR	0.80	0.55	0.05
	DS86/T65DR	1.06	1.11	6.40
Bladder	DS86	0.76	0.22	0.40
	T65DR	0.45	0.18	0.07
	DS86/T65DR	1.69	1.22	5.71
Ovary	DS86	0.74	0.16	0.39
	T65DR	0.40	0.12	0.08
	DS86/T65DR	1.85	1.33	4.88
Thyroid	DS86	0.85	0.41	0.43
	T65DR	0.70	0.45	0.04
	DS86/T65DR	1.21	0.91	10.75
Liver	DS86	0.76	0.29	0.39
	T65DR	0.47	0.18	0.08
	DS86/T65DR	1.62	1.61	4.88
Pancreas	DS86	0.72	0.18	0.42
	T65DR	0.40	0.12	0.08
	DS86/T65DR	1.80	1.50	5.25
Testis	DS86	0.78	0.32	0.38
	T65DR	0.65	0.40	0.05
	DS86/T65DR	1.20	0.80	7.60
Uterus	DS86	0.73	0.14	0.40
	T65DR	0.40	0.12	0.08
	DS86/T65DR	1.83	1.17	5.00

[a] The DS86 does not use constant transmission factors; these values are the averages from modelling the exposures of 19,113 individuals.

[b] These values are those given by Kerr (3), and are constants.

from reference Sh89

Table 4

Mean Shielded Kerma and Organ-Absorbed Dose (mGy) among Survivors Exposed to 0.01 Gy and Over

		Number of subjects	Total	Gamma	Neutron
Shielded kerma	DS86	41,719	295	287	8
	T65DR	41,316	414	350	64
Organ Dose					
Bone Marrow	DS86	40,701	242	239	3
	T65DR	41,316	219	201	18
Large intestine	DS86	39,859	223	222	1
	T65DR	31,958	198	186	12
Lung	DS86	40,382	240	238	2
	T65DR	41,316	194	180	14
Stomach	DS86	39,961	228	226	2
	T65DR	41,316	181	169	12
Female breast	DS86	25,252	240	236	4
	T65DR	25,211	309	276	33
Bladder	DS86	40,060	231	229	1
	T65DR	41,316	174	162	12
Ovary	DS86	24,581	211	210	1
	T65DR	19,563	190	181	9

from reference Sh89

Table 5

Mean Organ-Absorbed Dose Equivalent (mSv)

Site of Cancer	RBE	Mean Organ Dose Equivalent (mSv) DS86	 T65DR	DS86/T65D
Bone Marrow	1	242	219	1.11
	10	269	381	0.71
	20	299	561	0.53
Large Intestine	1	223	198	1.13
	10	232	306	0.76
	20	242	426	0.57
Lung	1	240	194	1.24
	10	258	320	0.81
	20	278	460	0.60
Stomach	1	228	181	1.26
	10	246	289	0.85
	20	266	409	0.65
Female Breast	1	240	309	0.77
	10	276	606	0.46
	20	316	936	0.34
Bladder	1	231	174	1.33
	10	239	282	0.85
	20	249	402	0.62
Ovary	1	211	190	1.11
	10	220	271	0.81
	20	230	361	0.64

Table 6

Factors Contributing To Uncertainty In A-Bomb Survivor Absorbed Dose*

Factors	Absorbed Dose Uncertainties	(FSD[%])
Yield	9.2	
Source Term	5.7	
Air Transport	10.1	
Survivor Coordinates	21.0	
Shielded Kerma	16.7	(11.8 - 35.5)
Organ Dose	7.3	
Total	31.5	(29.2 - 44.5)

* Data taken from reference Ka89

Figures 1 and 2

Comparison of DS86 and T65D Values

(from reference RE87)

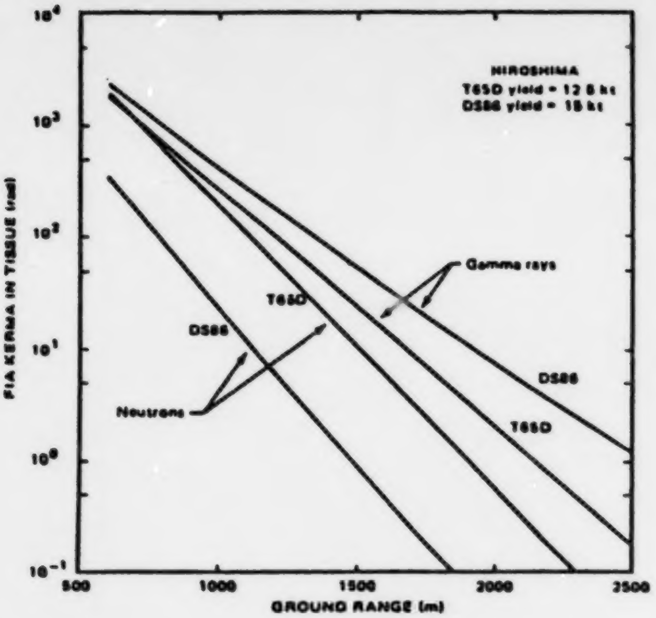

Figure 1. Comparison of DS86 and T65D values for the neutron and gamma-ray kerma in Hiroshima

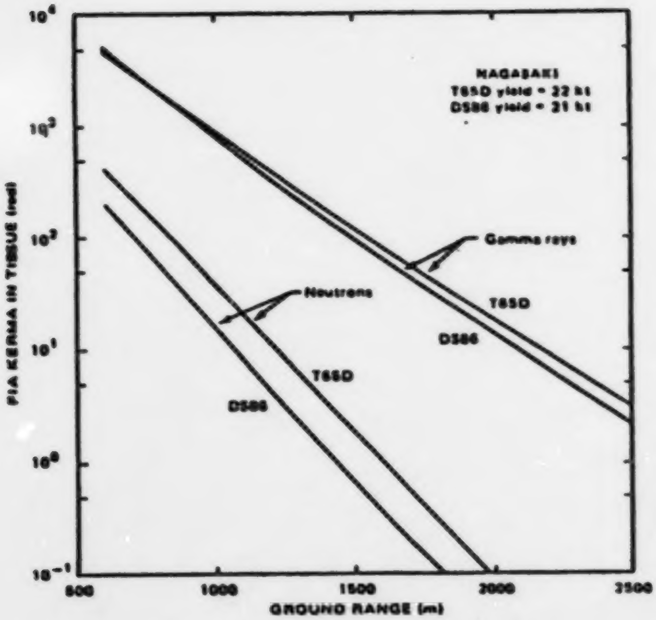

Figure 2. Comparison of DS86 and T65D values for the neutron and gamma-ray kerma in Nagasaki

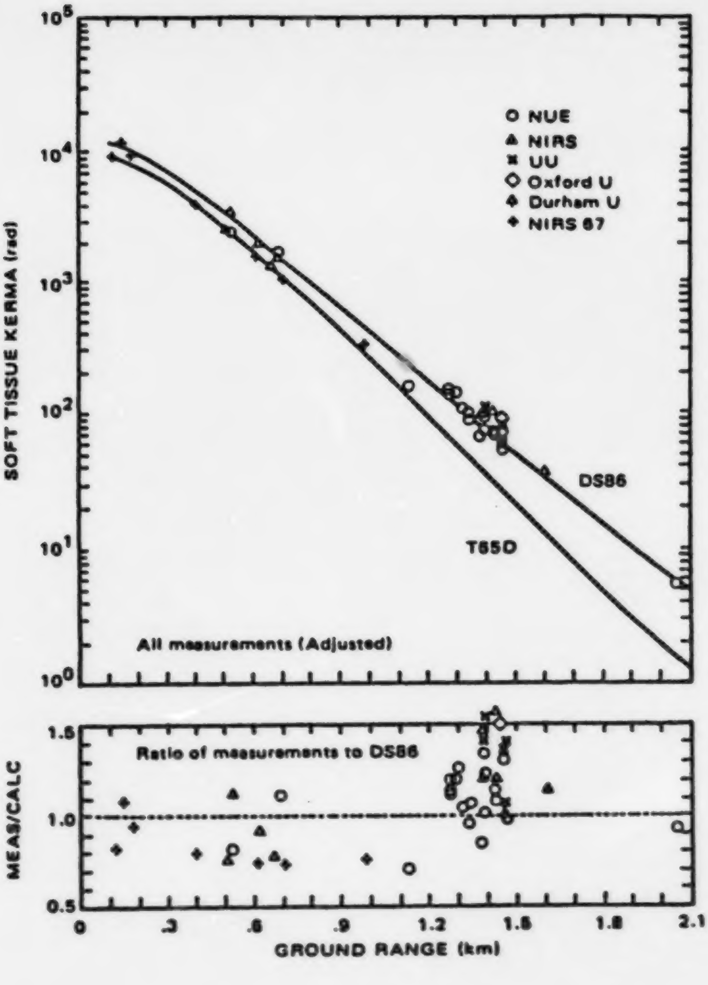

Figure 3: Comparison of theoretical calculations with thermoluminescence measurements on building materials exposed to gamma radiation at Hiroshima. Free-field kerma in soft tissue versus distance from hypocentre at Hiroshima (Appendix 4-11, from reference RE87).

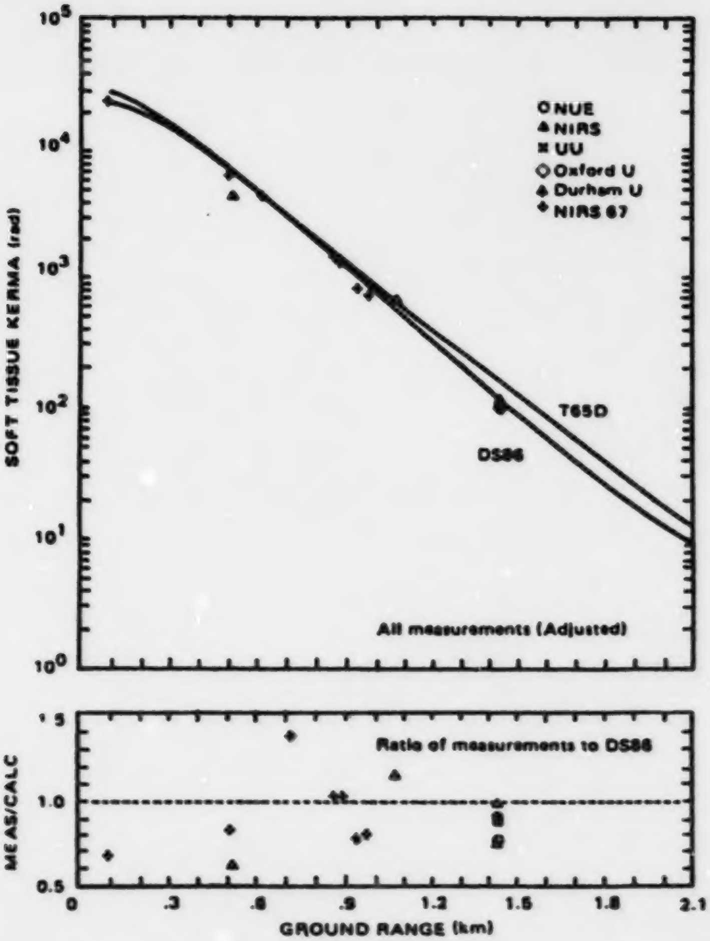

Figure 4: Comparison of theoretical calculations with thermoluminescence measurements on building materials exposed to gamma radiation at Nagasaki. Free-field kerma in soft tissue versus distance from hypocentre at Nagasaki (Appendix 4-11, from reference RE87).

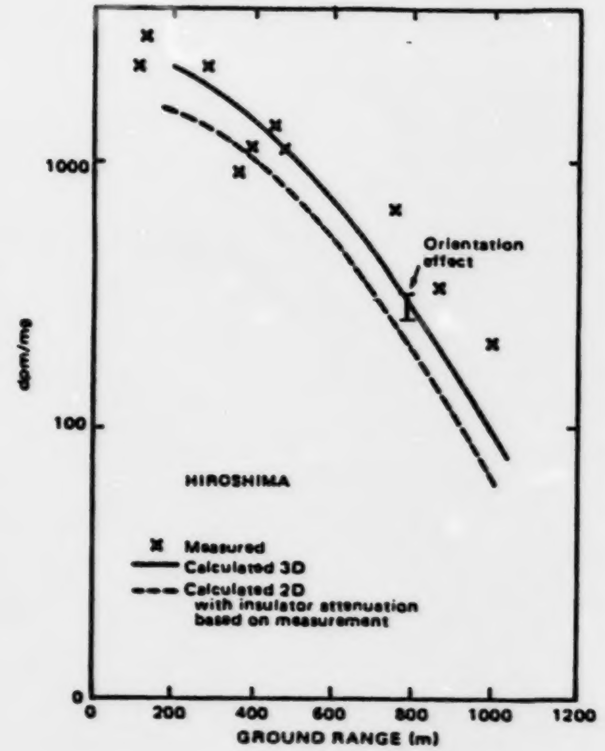

Figure 5: Sulfur activation at Hiroshima (taken from reference RE87). The measurements provide an estimate of exposures to high energy neutrons.

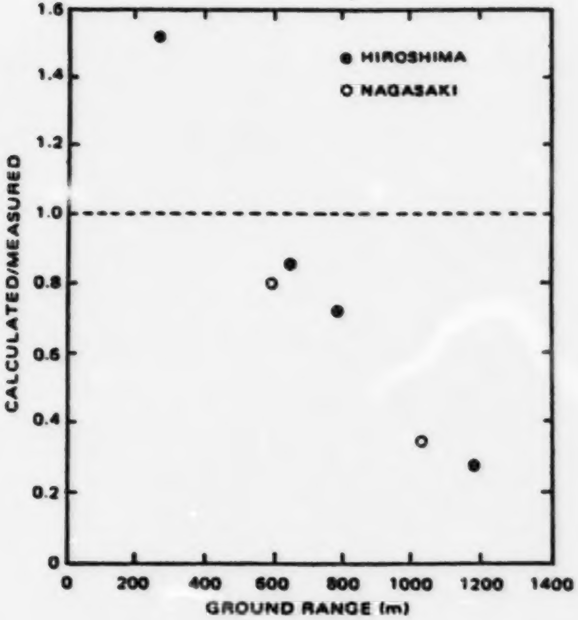

Figure 6: Comparison of measured and calculated ^{60}Co activities (taken from reference RE87). The measurements provide an estimate of exposures to thermal neutrons.

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ANNEX C

UNSCEAR 1988 Cancer Risk Estimates
(A.M. Marko)

In the 1988 UNSCEAR report (UN88) the subject of radiation-induced cancer was reviewed largely because of:

i) advances in knowledge of the biological mechanisms of the causation of cancers;
ii) the analysis of the dose-response relationship between cancers and radiation in the 1986 UNSCEAR report;
iii) the increased information from longer follow-up in the studies in Hiroshima and Nagasaki; and
iv) the revision of dosimetry in the atomic bombings of Hiroshima and Nagasaki.

The probability that a person exposed to radiation will develop cancer depends on several factors. Host factors, pertinent to the individual, are genetic back-ground, age, and sex. Other factors relate to the conditions of irradiation, the magnitude of dose, the time period over which the dose was delivered and quality of radiation. Other environmental factors, such as lifestyle and exposure to other toxic agents, can interact with radiation effects and influence the probability.

In the absence of detailed knowledge of the mechanism of radiation carcinogenesis, one must rely on epidemiological studies of humans exposed to radiation to estimate the possibility of contracting cancer. The most useful epidemiological studies are:

i) persons chronically exposed to high and intermediate doses of radiation when the risks were largely unknown;
ii) people who were chronically exposed to low doses of radiation occupationally, medically and environmentally;
iii) patients who received large doses of radiation to parts of their bodies therapeutically;
iv) patients who received exposures to radiation from medical diagnostic procedures;
v) survivors of the atomic bombings in Hiroshima and Nagasaki;
vi) survivors who received high doses of radiation because of an accident.

The methodology used to study the above groups is stated in the 1988 UNSCEAR report (UN88):

> "Two methods have been employed in the epidemiological investigation of the groups listed above: (a) cohort studies, in which exposed individuals are analyzed usually prospectively for their cancer experience compared with a suitably chosen non-exposed control group, and (b) case control studies in which individuals with cancer are matched with normal individuals of a control population and exposures are determined retrospectively. The first method has distinct advantages but, of course, can be employed only in special circumstances."

Only three major epidemiological studies with low-LET radiation were considered to provide risks of radiation-induced cancers in a variety of organs. These are:

i) more than 90,000 Japanese atomic bomb survivors, the Life Span Study (LSS) cohort. The new dosimetry has been estimated for a subcohort of about 76,000 subjects and a total of 5,936 cancers has been observed in this subcohort. Since there was no significant excess of cancers in survivors receiving less that 0.2 Gy, current risk estimates depend largely upon the 11,000-12,000 survivors who received more than 0.2 Gy in 1945.
ii) 14,000 largely male patients in the U.K. treated for ankylosing spondylitis with X-rays;
iii) 150,000 women in a number of countries treated for cervical cancer with X- or gamma-radiation.

The atomic bomb survivors are almost equally split into males and females and covered a full range of ages and doses. The other two studies deal with adults and treatment by radiation was a result of illness. All three studies comprise large number of people exposed to X- or gamma-radiation for short times but followed for long times - 40 years for the Life Span Study. In its final derivation of projected lifetime risk estimates, the 1988 UNSCEAR report used only the risk coefficients derived from the study of the Japanese bomb survivors.

The methods used to estimate life time projections of fatalities from radiation-induced cancers are summarized in Part II, para. 201, UNSCEAR 1988 report as follows:

> "Lifetime cancer experience is not yet available for any of the large epidemiological studies. Therefore, to project the overall cancer risk for an exposed population, it is necessary to use models that extrapolate over time data based on only a limited period of the lives of the individuals. Two such projection models have received particular attention: (a) the additive model which postulates that the annual excess risks arise after a period of latency and then remains constant, and (b) the multiplicative model in which the time distribution of the excess risk follows the same pattern as the time distribution of natural cancers, i.e., the excess (after latency) is given by a constant factor applied to the age-dependent incidence of natural cancers in the population."

The relative risk model is more likely to provide a better estimate of the life time projection of cancer risks for the Japanese bomb survivors.

Radiation at high doses is definitely carcinogenic but no cancers unique to radiation are induced. Leukemia (except for chronic lymphatic leukemia) is one of the prominent radiation-induced cancers followed by malignant tumours of the breast, thyroid, lung and other sites. Chronic lymphatic leukemia, squamous cell carcinoma of the cervix and Hodgkin's disease do not appear to be induced by radiation. The minimum latency period for leukemia is about 2 years and about 5-10 years for other tumours.

Japanese survivors in the Life Span Study have been followed for 40 years but another 40 years post-exposure is needed to determine the total risk to this population. However, the information on leukemia is probably now almost complete. Both in the Life Span Study and the ankylosing spondylitis study there was an initial rise in the leukemia absolute excess risk followed by a tailing-off in the risk. Nearly all the risk appears to have been expressed by 30 years after exposure.

For bone cancer induced by Ra-224 the temporal pattern of radiation-induced risk seems to be similar to that for leukemia. However, for cancers other than leukemia the excess risk in Japanese bomb survivors does appear to be relatively constant up to 40 years after exposure while in the ankylosing spondylitics, the uranium miners and the uterine cervical cancer studies there seems to be a tailing-off in the relative risk before this time.

In the 1986 UNSCEAR report (UN86) the Committee concluded that the dose-response relationship may be unique for each type of tumour, although it was still not possible to define precisely this relationship for many tumours. However, one general conclusion for low-LET radiations is that the shape of the dose-response curve is concave upwards, reaching a maximum and then declining. The decreased response at high doses of radiation is believed to be due to the killing of cells that are radiation-initiated from malignancy. The Committee also concluded in 1986 that carcinomas of the breast and possibly the thyroid have a linear dose response relationship at low and intermediate doses. Recent studies of 131-iodine administration to patients suggests that low doses are less effective in the production of tumours than acute doses of external radiation. Thus the dose-response relationship for thyroid cancers may be non-linear and also concave upwards as described above for other tumours. UNSCEAR 1988 (UN88) also expressed some doubts as to whether the dose response curve for induction of breast cancers was really linear.

The revised dosimetry at the two cities of Hiroshima and Nagasaki have largely eliminated an appreciable estimated neutron dose as a result of the atomic bombings. Because of this revision there is essentially no quantitative information on the biological effects of neutrons in man. Thus no useful estimate of the relative biological effectiveness (RBE) or quality factor for neutrons can be made from human data.

The way the Committee in the 1986 UNSCEAR report (UN86) uses subjective terms, such as "low", "intermediate" and "high" are illustrated in Table 1.

Table 1

Definitions of Low, Intermediate and High Doses and Dose Rates of Low-LET Radiation

	Low	Intermediate	High
Absorbed dose (in Gy)	< 0.2	0.2 - 2.0	2.0 - 10
Dose rate (in mGy/min)	<0.05	0.05 - 50	>50

Risk is defined as the probability of a harmful event, for example, a radiation-induced fatal cancer, and risk can be expressed as this probability per cent. The term "risk coefficient" is used in a general way to indicate the risk per unit dose (risk per gray in the case of absorbed dose or risk per sievert in the case of effective dose equivalent).

In the Life Span Study there is a statistically significant increase in the frequency of deaths with increasing dose for leukemia, multiple myeloma, and cancers of the oesophagus, stomach, colon, lung, breast, ovary, and urinary bladder. The risks for cancers of the oesophagus and urinary tract were only marginally increased. No significant increase is observed as yet for cancers of the gall bladder, pancreas, uterus and prostate or for malignant lymphoma. The study was extended to include other sites of cancer, such as bone, pharynx, nose and larynx and skin (except melanoma), but none of these sites showed a significant dose response relationship for the Japanese bomb survivors.

The Committee in its 1988 UNSCEAR (UN88) report estimated the cancer risk at doses of 1 Gy at high dose rate of low-LET radiation. The results are given in Table 2 which is taken from the 1988 UNSCEAR report.

The risks for 9 types of cancers have been assessed with some confidence. Also, the total risk from all types of cancers has been estimated. Two sets of numbers are given, one is derived from projections based on the additive (absolute) risk model and the other from projections based on the multiplicative (relative) risk model. The total cancer mortality risk coefficient for the average individual (also averaged over both sexes) is 4.5 x 10^{-2} per Gy on the additive risk model and 7.1 x 10^{-2} per Gy on the relative risk model based on the Japanese population. Similar values were calculated for the populations of the U.K. and Puerto Rico. Life-time risk coefficients based on age-specific risk coefficients for populations range from 4 x 10^{-2} Gy^{-1} (absolute risk model) to 11 x 10^{-2} Gy^{-1} (relative risk model). For the working population (aged 25-64 years) the additive risk model yields a risk coefficient of 4 x 10^{-2} Gy^{-1} while the multiplicative model gives a value of 8 x 10^{-2} Gy^{-1}

The Committee agreed that there was a need for a reduction factor to modify these risks for low doses and low dose rates. They believed this dose reduction effectiveness factor (DREF)[1] to be between 2 and 10.

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[1] DREF: a factor used to derive risk estimates of exposure to low-LET radiation at low dose rates, from risk estimates calculated from data associated with exposures at high dose rates.

Table 2

(Taken from Table 9 of 1988 UNSCEAR Report)

Per caput lifetime excess cancer deaths probability following exposure to 1 Gy organ absorbed dose at high dose rate of low-LET radiation (per cent)

(based on the population of Japan using an average age risk coefficient)

	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	0.97	0.93
All cancers except leukemia	6.1	3.6
Bladder	0.39	0.23
Breast[a]	0.6	0.43
Colon	0.79	0.29
Lung	1.5	0.59
Multiple myeloma	0.22	0.09
Ovary[a]	0.31	0.26
Oesophagus	0.34	0.16
Stomach	1.3	0.86
Remainder	1.1	1.0
Total	7.1	4.5

[a] The data for breast and ovary apply to females only. Therefore the value has to be divided by 2 to apply for populations composed of equal numbers of both sexes.

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ANNEX D

BEIR V 1990 Cancer Risk Estimates

(G.R. Howe)

INTRODUCTION

The Report of the 5th National Research Council's Committee on the Biological Effects of Ionizing Radiation (BEIR V) was published in January 1990. The report focuses on external low-LET radiation and its health effects. A previous report in this series (BEIR IV) was concerned with the health effects of exposure to radon and other internally deposited alpha-emitters. A major part of the BEIR V Report is concerned with cancer risk, and differs in three major respects from the previous BEIR Report which dealt with low-LET radiation (BEIR III), which was published in 1980. The three major differences are:

1. The Committee carried out fairly extensive statistical analyses of the original data from a number of epidemiologic studies of human populations exposed to ionizing radiation, in order to arrive at risk estimates.

2. Statistical modelling techniques were used to analyze those data which allowed for modifying the effects upon risk of related factors such as age at exposure, time since exposure, and age at risk. Thus, such models differ from the usual simple constant relative risk or constant additive risk models which traditionally have been used, in particular in the BEIR III report and the 1988 UNSCEAR Report.

3. New doses (DS86) have been estimated for members of the cohort of survivors of the atomic bombings at Hiroshima and Nagasaki. These estimates are generally lower for both populations than the previously utilized T65 doses when expressed in dose equivalents with an appropriate correction factor for the RBE of neutrons. The estimated neutron exposure at Hiroshima has now been reduced to negligible levels. These new doses, expressed in sieverts, have been used in the models analyzed by the BEIR V Committee, and this has a substantial impact on lifetime risk estimates. The absence of a large neutron component means that there is essentially no human data relating cancer risk to exposure to neutrons.

Separate analyses were conducted by the BEIR V Committee for a number of specific cancer sites. For many of the cancer sites the number of observed deaths was too small to produce reliable risk estimates. Therefore, in order to estimate lifetime excess cancer mortality risks, the Committee developed preferred models separately for leukemia, respiratory tract, breast, digestive organs, and all other cancers combined. A life-table method was then used to estimate the increase in mortality from all cancers following various exposure scenarios by adding the individual risks for the groups of cancers considered. Monte Carlo simulations were used to estimate approximate confidence intervals.

Modelling Results

The models fitted to the various data sets compared the fit of the linear with the linear quadratic dose-response relationship, and the modifying effects upon risk of sex, age at exposure, time since exposure, and age at risk. The data set which contributed most to the risk estimation procedure was that for the atomic bomb survivors, for which data were available for all of the cancer sites discussed below. For some of the sites, particularly breast, other data sets were incorporated into the analysis. The breast cancer data included the Canadian Fluoroscopy Study, and the studies of women treated in New York State for postpartum mastitis, and in Massachusetts for tuberculosis. Wherever possible, results were compared with those for the ankylosing spondylitic study, though no formal joint analysis was carried out. In general, the linear relative risk model proved to be the preferred model, with modification by sex and time-dependent factors which varied with the particular site being considered. Only for leukemia was the preferred dose-response a linear quadratic model. The constant additive risk model did not provide a good fit for any of the cancer sites examined, and the Committee concluded that this model was no longer tenable. These results contrast with the conclusions of the BEIR III Committee, where the preferred dose-response relationship was generally linear quadratic, and both constant relative and constant additive risk models were presented. Further details for each of the sites or groupings considered for the combined mortality analysis are now discussed specifically.

Leukemia

A minimal latency period of two years was assumed for this model. The best fit was provided by a relative risk model in which the dose-response relationship was linear quadratic. There is a distinct difference in risk for those exposed under the age of 20, compared with those past that age, with the two groups having different dependencies on time since exposure. Within each of these two groupings, however, the specific age at exposure made little difference to risk. The Committee suggested that if a continuous, rather than a step function, is preferred for purposes such as setting probabilities of causation, averaging techniques may be used to fit the age at exposure effect.

The Japanese survivors of the atomic bombing of Hiroshima and Nagasaki (LSS) are the major source of human data. However, data collection was only begun in 1950 and hence there is no information in the first five years after exposure. This is particularly relevant for leukemia where the latent period may be as short as two years. Based on data from spondylitics and cervical cancer patients one could reasonably argue that nearly one-half of the excess leukemias would have been observed within the first 5 years after exposure. The BEIR V Committee accounted for the 2 to 5 year post exposure period by extrapolating to two years the excess relative risk observed for the 5 to 10 year post-exposure period. This resulted in an approximately 15% increase in the lifetime risks. The Committee cautioned, however, that this procedure may lead to an underestimation of the leukemia risk which should be borne in mind when interpreting their risk estimates (BE90).

Respiratory Cancers

A minimal latency period of 10 years was assumed for respiratory cancers. The choice of relative, rather than additive risk model was based essentially upon the rather more parsimonious nature of the former model. The preferred dose-response was a simple linear model, and there are modifying factors, due both to sex and time since exposure. Women had a somewhat higher relative risk than men, and the time since exposure coefficient had a negative value, which implies that relative risk decreases with time since exposure. Although a constant relative risk model (i.e. no time dependency) yielded an approximately equally good fit to the data, the Committee preferred the inclusion of a time dependent term, since a similar pattern has been seen in the ankylosing spondylitics data. Age at exposure had virtually no effect on the fit of the model, though the Committee pointed out that little data are yet available for respiratory cancer on those exposed at very young ages in the Atomic Bomb Survivors' Study. The BEIR V Committee did not refer to the RERF report (Sh88) which indicated that smoking and radiation acted additively rather than multiplicatively in lung cancer induction.

Breast Cancer

For breast cancer, a minimal latent period of five years was used, based on the data from the several available series. Again, as for respiratory cancer, a linear relative risk model was preferred. The most notable effect on risk was due to age at exposure, with much higher risks being seen with those exposed before age 15, and with a sharp reduction in risk for increasing ages at exposure, and a very low risk for those exposed past the age of 40. Two specific models were developed, one for those exposed under age 15, and one for those exposed past this age. For both series, there was a linear quadratic dependency on time (using log time rather than time itself), which indicates a peaking of effect after some 15-20 years, followed by a decline in the relative risk. This model was, of course, restricted to women, in view of the absence of any data for male breast cancer.

Digestive Organ Cancers

A minimal latent period of ten years was used for this grouping. Again the preferred model was a linear relative risk one, which was substantially modified by age at exposure, with much greater risks being seen for those exposed under the age of 30, compared to those exposed past this age. Women appeared to have somewhat greater relative risk than men, though there was no evidence of any time dependency for these sites.

Other Cancers

Again, a minimal latency period of ten years was used for this group of solid tumours. A simple linear relative risk model with decreasing risk with increasing age at exposure was the preferred model. There appear to be no effects of sex or time since exposure.

Excess Cancer Mortality Estimates

The BEIR V Committee used the relative risk models described above for leukemia, respiratory cancers, breast cancer, digestive organ cancer, and other cancers combined to estimate the excess total cancer mortality following various exposure scenarios. The base rates used were the mortality rates for the United States for 1979-81. Table 1 compares the projected risks as given in BEIR V with those given in BEIR III. The differences for the non-leukaemic cancers using the relative risk model are driven partly by the change in dosimetry for the A-bomb Survivors' Study, partly by the negative time dependency seen for breast and lung in the preferred BEIR V models, and partly by the lack of a dose rate effectiveness factor in the BEIR V estimates. It should be noted that the confidence bounds provided elsewhere in the BEIR V Report probably represent the most effective estimates of such bounds, computed by any of the summary reports published to date, but these estimates are dependant on the particular risk projection models chosen.

Table 1

Comparison of Lifetime Excess Cancer Risk Estimates for the General Population from the BEIR III and BEIR V Reports

	Cancer deaths per 10 000 persons exposed to a single dose of 0.1 Gy of low-LET radiation
BEIR III	
Additive risk model	0.95 - 7.7 - 16.7 (a)
Relative risk model	2.8 - 22.6 - 50.1 (a)
BEIR V	79 (b)

(a) Data from Tables V-1 and V-2 of BEIR III. The three values in each row represent numbers derived assuming a quadratic, linear-quadratic and linear dose-response relationship for low-LET radiation. The majority but not all members of the BEIR III Committee favoured the linear-quadratic model.

(b) Data from Table 4-2 of BEIR V. Estimates for solid cancer derived using a linear dose-response model, without application of a dose rate effectiveness factor as recommended elsewhere in BEIR V.

Dose Rate Effectiveness Factor

The BEIR V Committee considered the issue of the effect of dose rate and fractionation on risk, and also the possible reduction in risk per unit of exposure at very low doses. They concluded that little human evidence was available to address these topics. The most relevant evidence probably comes from the breast cancer data where risks are quite similar for A-bomb survivors who received unfractionated doses at very high rates, for women treated by radiotherapy for postpartum mastitis where exposure was given in several fractions, and for female fluoroscopy patients where doses were substantially fractionated, and generally given at lower dose rates. However, these latter examples are not directly comparable to the very low doses and dose rates which are more relevant to the current situation, so although these data provide little evidence of the dose rate effect, this interpretation must be substantially limited. The BEIR V Committee concluded that a dose rate effectiveness factor of 4-5 times for low-LET radiation was not unreasonable, based on non-epidemiologic data. Similarly, they state that at very low doses the effect per unit dose might be reduced by a factor of 2 or more. It should be noted, of course, that the leukemia risk model is an exception, in that the linear quadratic form of the dose-response relationship de facto introduces a dose rate effectiveness factor of approximately 2.

The BEIR V Committee also pointed out that human data cannot exclude the possibility of a threshold effect, i.e., zero risk at very low doses. However, it should be noted that the Committee does not offer support for the existence of a threshold effect, and the statistical models used for risk estimation in the report do not allow for a threshold effect, but assume that any dose leads to an increase in risk.

References

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BE88	BEIR IV Report. Health effects of radon and other internally deposited alpha-emitters. Committee on the biological effects of ionizing radiations. National Academy of Sciences, Washington (1988).
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ANNEX E

The Effect of Dose Rate on Risk Estimates for Low-LET Radiation

(D.J. Gorman)

The models used to derive risk estimates are based primarily on data from high exposures - in the case of the A-bomb survivors a single instantaneous exposure, and in the case of most of the medical exposures, fractionated but high dose rate exposures. The application of these models to low dose-rate exposures (<0.05 Sv/y) requires consideration of a dose rate effectiveness factor (DREF)[1].

The existence of a dose-rate effect for low-LET radiation has been widely demonstrated in experimental cellular and animal systems and, in general, the effectiveness of a given dose tends to decrease with decreasing dose rate. In the case of low-LET radiations, the reduced effectiveness of a dose delivered at low dose rates is a consequence of factors such as the repair of initial damage during a protracted exposure. In the case of high-LET radiations, the dose rate effect is much reduced, primarily because of the reduction in the effectiveness of the repair mechanisms. There is also some evidence that for higher doses of high-LET radiations, protracting an exposure may lead to an increase in the induction of cancer and mutations (BE90).

NCRP Report 64 (NC80) provides a comprehensive summary of the literature on DREFs related to hereditary and carcinogenic effects. The report concludes that a DREF of between 2 and 10 could be rationalized on the basis of radiobiological data when the actual absorbed dose is 0.2 Gy or less, or the dose rate is 0.05 Gy per year or less (NC80). The data in NCRP-64 have been considered in numerous recent reports concerned with risk estimates for the induction of cancer by radiation (UN86, UN88, BE90, IC90). The resulting dose rate reduction factors, as summarized in the BEIR V report, are given in Table 1.

Since NCRP 64 is the major reference upon which later committees have based their selection of a DREF to be applied to humans, it is important to review the assumptions upon which this recommendation is based. In reviewing this document it is important to note the Committee's statement:

"There are no human data that allow definitive statements with respect to whether a dose-rate influence applies, or does not apply. There are sets of data that have been taken to indicate that there is a dose-rate effect. The same data have also been used to support the claim that there is not a dose-rate effect" (NC80: p157).

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[1] DREF: a factor used to derive risk estimates of exposure to low-LET radiation at low dose rates, from risk estimates calculated from data associated with exposures at high dose rates.

The recommendation of NCRP-64 that a DREF should be applied to humans is based on the following reasoning by the NCRP Committee:

1. The dose-rate dependence is so well demonstrated across species and endpoints that it is difficult to believe that man would be a single exception.

2. The human data available does not exclude the presence of a dose rate effect.

3. Therefore a DREF should be assumed to apply to carcinogenesis in humans. This factor is likely to be between 2 and 10.

The UNSCEAR Committee (UN86, UN88) based their review of the data on dose rate dependence on NCRP-64 and concluded that "the carcinogenic effects of low-LET radiation are generally smaller at low doses and at low dose rates compared with those at high doses and dose rates. The reduction factor will vary with dose and dose rate and with organ system, but will generally fall within the range 2 to 10" (UN88, p.492, para. 607). UNSCEAR (UN86, p.243, para 486) also concluded that "for sparsely-ionizing radiation, linear extrapolation from about 2 Gy down would not overestimate the risk of breast and possibly thyroid cancer. It would slightly overestimate the risk of leukemia, and it would definitely overestimate the risk of bone sarcoma. For lung cancer, lack of direct evidence does not permit any estimate to be made of the magnitude of the overestimate".

The National Radiological Protection Board in the United Kingdom (NR88) has recommended the use of a DREF of 3 for all cancers except breast, for which a DREF of 2 is recommended. The rationale for the value 3 is that (1) it is the median value of the DREFs found in a large number of animal experiments, (2) it is consistent with the value of 3 used by UNSCEAR and ICRP for assessing the risk of genetic damage at low doses, and (3) it is supported by the information on thyroid cancer induction resulting from exposure to external radiation or intakes of I-131. The factor of 2 is chosen for breast in the absence of data directly showing a dose rate effect in humans.

Although the BEIR V report provided estimates of DREF values (Table 1), it did not recommend any particular value for induction of cancers other than leukemia by low-LET radiation. The BEIR V risk estimate for leukemia includes an implicit DREF of 2.1. The executive summary of the BEIR V report provides a risk estimate for death from cancer following an acute dose of 0.1 Gy of low-LET radiation of 0.8%. The report indicates that "for low-LET radiation, accumulation of the same dose over weeks or months, however, is expected to reduce the lifetime risk appreciably, possibly by a factor of 2 or more".

The values of the DREF given for human leukemia in the BEIR V report and in the BEIR III report arise from the use of a linear-quadratic dose response model. In such a model, the linear term predominates at low doses, and presumably at low dose rates while the quadratic term predominates at high doses and dose rates. If both linear and linear quadratic models are fitted to the data, the ratio of the linear coefficients of the two models yields an estimate of the DREF. The BEIR V report, in using a linear quadratic model for leukemia, implicitly applied a DREF of 2.1 to the leukemia risk estimate.

For all other cancers a linear model was used with no DREF. The BEIR III Committee, on the other hand, estimated a DREF from the leukemia data and applied this as a fixed constant to the non-leukemia data (BE90 p.174).

The DREFs estimated in NCRP 64 and applied in subsequent reports discussed above are based on life shortening, carcinogenesis and metagenesis in laboratory animals. The primary issue to be addressed is the application of this data to humans in the absence of any direct human evidence for a DREF.

The most recent human data from the Japanese Life Span Study (LSS), as analyzed by UNSCEAR, show a non-linear dose response relationship for leukemia, where the relative risk for those exposed at greater than 0.5 Gy is twice that of those exposed at less than 0.5 Gy (UN88, p.491. para 603). Shimizu et al. (Sh90), in analyzing the data, found a DREF of 1.99 for leukemia and 1.15 for all other cancers. The data for all solid cancers combined fit more closely to a linear dose-response relationship.

In one experimental mammary tumour system in rats, irradiation with X-rays and gamma-rays produced a linear-dose response with little fractionation and dose-rate dependence (UN86, p.14, para 54). However, other data suggest the possibility of a non-linear dose response for appearance of mammary carcinomas in rats after exposure to low-LET radiation, and indicate no excess carcinomas with gamma-rays at low dose rate at least for doses up to 1 Gy (Jo89, Br90).

With respect to thyroid cancer, two human studies provide evidence for a strongly linear dose-response relationship with no dose-fractionation effect (UN86, p.226). However, there is also evidence that I-131 deposited in the thyroid gland is about one-third to one-quarter as effective as external radiation at high dose rates (UN88, p.491). There is some dispute as to whether this is as a result of a dose rate effect or due to non-uniformity of dose distribution in the gland.

Human data, from the LSS study, suggest that a DREF of about 2 or more for leukemia may be appropriate. There is no good human evidence to either support or disprove a dose rate effect for other organs (UN88, p.491-92).

Problems Inherent in the Use of a DREF

There are some practical problems in applying a dose reduction factor to the risk estimates for humans. The DREF is applicable only to low-LET radiation. The variations in the age-specific risks are of the same magnitude as the variation in the DREF (see Table 2). The risk factors derived in the BEIR V report are averages over a population with the same age distribution as the U.S population. They therefore represent the best estimate of risk to that population and not to individuals in that population. That portion of the individual risk solely attributable to age may vary by a large factor from the average population risk. Where radiation protection standards are applied to the protection of individuals, possible variations in individual DREF values should also be considered.

Conclusion

Major international bodies which have recently reviewed the data on radiation effects (UN88,NC80,BE90,IC90) all agree that a DREF should be applied when extrapolating risks calculated at high doses and dose rates to low doses and dose rates. UNSCEAR, BEIR, and NCRP all agree on a range of DREFs of 2 to 10, with BEIR V suggesting a best estimate of 4-5. A conservative value would be 2, which is that recommended by ICRP.

A degree of caution should be exercised when applying a DREF to human risk estimates. Although there is ample evidence for such an effect in cellular and animal systems, there is very little supporting human data. Recent evidence from the life span study of the Japanese A-bomb survivors indicates a dose effect for leukemia of about 2 or more. There is some human evidence both for and against the presence of a dose rate effect for cancers of the breast and thyroid. There is no human evidence for or against a dose rate effect for other cancers.

The ICRP concept of a "nominal risk coefficient" for defined populations - the general population and the working population - is useful. It must be borne in mind, however, that this value is an average over these populations and should not be applied to individuals. When assessing risk to individuals, consideration should also be given to age-specific risk coefficients such as those in the BEIR V report (see Table 2).

Table 1
(FROM BEIR V)

Summary of Dose-Rate Effectiveness Factors for Low-LET Radiation

Source of Data	Observed Full Range of Values	Limited for Narrow Range of Values	Single Best Estimate
Human leukemia			
BEIR V	-	-	2.1
BEIR III	-	-	2.0 to 2.5
Laboratory animal studies			
Specific locus mutation	3-10	3-7	5
Reciprocal translocation	5-10	5-7	5
Life shortening	3-10	3-5	4
Tumorigenesis	2-10	2-5	4

NOTES:

1. BEIR V and BEIR III leukemia estimates arise from the application of a linear-quadratic dose-response model.

2. The narrow range of values recognizes that the upper limit of the full range values may include some experimental conditions that are not entirely relevant.

3. Single best estimates are appropriate for all low dose-rate, low-LET radiation exposures delivered intermittently, or even continuously, over periods of months to years.

Table 2
(FROM BEIR V)

Cancer Excess Mortality by Age at Exposure and Site for 100,000 Persons of Each Age Exposed to 0.1 Sv (10 rem)

MALES

Age at Exposure	Total	Leukemia	Non-Leukemia	Respiratory	Digestive	Other
5	1,276	111	1,165	17	361	787
15	1,144	109	1,035	54	369	612
25	921	36	885	124	389	372
35	566	62	504	243	28	233
45	600	108	492	353	22	117
55	616	166	450	393	15	42
65	481	191	290	272	11	7
75	258	165	93	90	5	--
85	110	96	14	17	--	--
Average[a]	770	110	660	190	170	300

FEMALES

Age at Exposure	Total	Leukemia	Non-leukemia	Breast	Respiratory	Digestive	Other
5	1,532	75	1,457	129	48	655	625
15	1,566	72	1,494	295	70	653	476
25	1,178	29	1,149	52	125	679	293
35	557	46	511	43	208	73	187
45	541	73	468	20	277	71	100
55	505	117	388	6	273	64	45
65	386	146	240	--	172	52	16
75	227	127	100	--	72	26	3
85	90	73	17	--	15	4	--
Average	810	80	730	70	150	290	220

[a] Averages are weighted for the age distribution in a stationary population having U.S. mortality rates. See also footnotes to Table 4-2 (BE90). 90% confidence interval for these risk estimates are listed in Annex 4D, Table 4D-4 (BE90); for example, the 90% confidence intervals on the lifetime risk estimates for excess leukemias in males age 5 at exposure range from 20 to 455 using the model preferred by the BEIR V committee. Alternative models considered in Table 4D-6 of this report (BE90) give appreciably smaller risk estimates for leukemia than those listed above.

References

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UN77	United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Genetic Effects of Ionizing Radiation. pp 425-564 in Sources and Effects of Ionizing Radiation. UNSCEAR Report (1987).
UN86	United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Genetic Effects of Radiation. pp 7-164 in Genetic and Somatic Effects of Ionizing Radiation. UNSCEAR Report (1986).
UN88	United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). Sources, Effects and Risks of Ionizing Radiation. UNSCEAR Report (1988).

ANNEX F

Prenatal Risk Estimates

(K.L. Gordon)

INTRODUCTION

It has long been recognized that the mammalian embryo and foetus has a much higher degree of radiosensitivity than the adult or young of the species. From a radiobiological standpoint, this finding is to be expected because cells that are actively dividing are more radiosensitive than those that only divide occasionally, or cannot divide. Given the number of proliferating cell populations in the embryo or foetus, it follows that their tissues are especially prone to radiation injury.

Effects of prenatal irradiation may be classified as "short-term", "long-term" and "late". Short-term effects are those which manifest at the cellular or tissue level or both. When the initial damage is sufficiently great, it may result in malformation or spontaneous abortion (miscarriage). Long-term effects are often biologically-modified expressions of previously induced, and sometimes latent defects. They include reduced organ and body growth, impaired cell maturation, as well as structural and functional defects, especially of the central nervous system. The potential late effects of intrauterine irradiation include carcinogenesis and hereditary damage.

Much of the evidence concerning these effects comes from extensive studies of different animal species (primarily rodents) experimentally irradiated in utero, under highly controlled circumstances, to examine the effect of radiation quality, dose, dose rate, temporal dose distribution compared with gestational age, etc. Unique radiation effects seen in experimentally irradiated animals include gross structural malformations, growth retardation, death of the embryo or foetus, and central nervous system abnormalities. The animal data evidence with respect to carcinogenesis is equivocal and "does not convey an overall impression of a significantly higher susceptibility to the induction of tumours of animals irradiated in utero" (UN86).

Certain groups of prenatally exposed human populations (with considerably less accurate dosimetry and gestational staging) have also been followed closely. The populations most extensively studied over the longest period are those individuals irradiated in utero during the atom bomb explosions at Hiroshima and Nagasaki. Other groups studied in the U.K and the United States received prenatal radiation exposure for medical purposes. In humans, damage to the central nervous system manifesting as severe mental retardation or diminished intelligence is the best documented of the developmental abnormalities following prenatal radiation exposure. With respect to stochastic effects, the correlation between in utero exposure and an increased risk of cancer in childhood and later life is not as clear-cut. While the data are inconclusive, international scientific advisory bodies currently accept a causative link between prenatal radiation exposure and cancer (UN86, UN88, BE90, IC90). This link is assumed simply on account of prudence for regulatory purposes.

THE MAIN PHASES OF PRENATAL DEVELOPMENT

Pre-Implantation: This period commences with fertilization of the ovum by means of penetration and activation by the spermatozoon. Mingling of chromosomes during the fusion of the haploid pronuclei of the sperm and ovum converts the fertilized ovum into a zygote. The zygote is a large diploid cell that is the beginning or primordium of a human being. As the zygote passes along the fallopian tube to the uterus, cleavage (rapid mitotic cell divisions) of the zygote into smaller cells occurs. The new cells, known as blastomeres, become smaller with each division. After several divisions, a mulberry-shaped mass (known as the morula) of 16 blastomeres forms. When the morula enters the uterus, fluid passes into the intercellular spaces between the blastomeres, and gradually the spaces become confluent, forming a large (blastocytic) cavity. At this point (about four to five days after fertilization), the developing embryo becomes known as a blastocyte. The blastocyte attaches itself to the lining of the uterus where it subsequently becomes embedded (implantation).

Embryonic Period: In humans, the actual implantation takes several days and the whole process (from the moment of conception (fertilization) until full implantation with placental blood flow established) is complete by about the 14th day (BR81). The process of germ layer formation (gastrulation) begins at the end of the first week and is completed during the third week, corresponding to the beginning of embryogenesis (formation of the embryo). During gastrulation the embryonic disc which initially formed in the blastocyte develops into three layers of cells known as the germinal layers (ectoderm, mesoderm and endoderm).

Masses of cells from the three germinal layers later split into smaller groups of cells which eventually result in the production of specific organs and tissues. The embryonic ectoderm gives rise to the epidermis, nervous system, sensory epithelium of the eye, ear and nose, as well as tooth enamel. Embryonic endoderm forms the linings of the digestive and respiratory tracts. The embryonic mesoderm becomes muscle, connective tissue, bone and blood vessels (Mo88).

The fourth to eighth week constitutes a very important period of embryonic development because all major external and internal structures begin to appear during these five weeks (organogenesis). By the end of the eight week, all the main organ systems have begun to develop, but the function of most organs is minimal. It is estimated that by the end of the embryonic period (eight full weeks after fertilization), the human embryo (crown-rump measuring about 30 mm and weighing 2-2.7 g) already possesses greater than 90% of the more than 4500 structures present in the adult body (UN88).

Foetal period: The transition from embryo to foetus is neither abrupt or spectacular, but the name change is made to signify that the embryo has developed unmistakable human characteristics. Growth, differentiation, development and maturation of body organs and systems proceed during the foetal period until parturition (birth). In humans the foetal period commences at the beginning of the ninth week after fertilization and continues through to birth at approximately 266 days or 38 weeks after conception.

Development of the Central Nervous System (CNS): The development of the central nervous system is outlined in greater detail below. At the gestational age of:

0-7 weeks, the precursors of the neurons and neuroglia (the two principal types of cell that give rise to the cerebrum) have emerged and are mitotically active;

8-15 weeks, a rapid increase in the number of neurons occurs; they migrate to their ultimate developmental sites and lose their capacity to divide, becoming perennial cells;

16-25 weeks, differentiation in situ accelerates, synaptogenesis that began about the 8th week increases, and the definitive cytoarchitecture of the brain unfolds;

26 or more weeks after fertilization, there is continued architectural and cellular differentiation and synaptogenesis of the cerebrum current with accelerated growth and development of the cerebellum.

Development of the human brain differs from the development of most other organs or organ systems (IC86).

(a) Structurally, the brain is one of the most complex organs of the body, with an involved architecture in which different functions are located in different structures. The pattern of CNS differentiation and growth is quite complex, taking place for various structures at different times and durations. Inactivation of any centre in the brain will result in a deficiency or loss of its specific functions. This is particularly true of the neocortex, which develops over a long period of time;

(b) Organizationally, brain function critically depends on the disposition and interconnection of structures and cells. Developmentally, normal structure and function hinge on an orderly sequence of events (cell division; programmed cell death; migration, including the positioning and selective aggregation of cells of the same kind; differentiation, with the acquisition of new cell membrane properties; and synaptic interconnection) each of which must occur correctly in time and space. Both cooperation and competition are involved in many of these events; and

(c) Functionally, the neurons of the central nervous system are not self-renewing, but are generated once and for all in the course of cortical histogenesis. Neuronal loss in humans is irreparable because repair through re-population cannot occur. The capacity of neuronal precursors to divide is exhausted during the histogenesis of the cerebral cortex and culminates in differentiated neurons which do not divide. Inactivation of the neurons or their precursors by the action of radiation or any other embryotoxic agent is likely to result in permanent loss of mental functions (UN86, UN90).

Congenital Anomalies

The process of development from conception to birth is highly complex, and in many cases unsuccessful. While the rate of human embryonic loss between conception and recognized pregnancy is not precisely known, it is generally agreed that loss is very high, about 40% between conception and 20 weeks of pregnancy, with 10% occurring between 10 and 18 weeks. Most such spontaneous abortions (miscarriages) occur before pregnancy has been diagnosed, and often before the first missed period. About 15% of recognized conceptions will lead to spontaneous abortion during early and intermediate foetal development, and one-third of these aborted foetuses carry some kind of chromosomal aberration. The occurrence of morphological abnormalities in the spontaneously aborted foetuses is also much higher than that seen in newborns, pointing to the role of foetal death in selecting morphologically and chromosomally abnormal products of conception (UN86).

Therefore, the 6% of live-born children who have abnormalities (that are sufficiently severe to disturb their physical well being and in some cases to decrease their viability) are only the survivors of a much greater number of malformed or otherwise defective embryos or foetuses that have died at some stage of prenatal development. The estimate of incidence of congenital abnormalities climbs to about 8.5% when less serious abnormalities (i.e. inguinal or umbilical hernias, minor structural blemishes or conditions of little medical significance), low birth weight, and other congenital handicaps not always ascertained at birth are included (UN86).

Based on their aetiology, congenital abnormalities at birth are subdivided into those due to:

(a) mutant major genes, autosomal or gonosomal, recessive or dominant, representing 6% of the total prevalence;
(b) multifactorial causation; polygenic disposition interacting with a variety of mostly unknown environmental factors, which account for 50% of the total;
(c) chromosomal aberrations, which contribute 5% of all cases;
(d) environmental factors including maternal diseases or infections, maternal nutrition, tobacco or ethanol use, pharmaceuticals, exposure to chemicals, ionizing radiation and other possibly teratogenic agents; representing 6% of the total; and
(e) those where cause is unknown at present, accounting for about 30% of all cases.

Several recent Canadian epidemiological studies (based on the British Columbia Health Surveillance Registry) of genetic disorders by Anderson and Baird et al (An87, Ba88) conclude that before an approximately age of 25 years, 53/1,000 or 5.3% of live-born individuals can be expected to have genetic or partially genetic diseases. This group represents a total estimate of genetic load, consisting of single-gene disorders are which responsible for 3.6/1,000 cases; chromosomal disorders responsible for 1.8/1,000 cases; and multifactorial disorders responsible for 46.6/1,000 cases. If all congenital abnormalities (including those without a genetic component) ascertained at birth or expressed before age 25 are considered, the prevalence rises to 79.4/1,000.

Teratogens: Human teratogens are environmental agents that result in an increased incidence of congenital malformations after conception. Known human teratogens include drugs (e.g. ethyl alcohol, thalidomide), chemicals (e.g. methylmercury), infectious agents (e.g. rubella virus, cytomegalovirus) and high levels of ionizing radiation. Teratogens present during the first two weeks of development either have no teratogenic action or their effects are so severe that they cause the death and spontaneous abortion (miscarriage) of the embryo. The stage of development of the embryo or foetus determines its susceptibility to teratogens. When the embryo's parts and organs are forming (during the organo-genetic period, particularly from days 15 to 60), teratogenic agents may cause major congenital malformations. Although the critical period for brain growth and development is from the third to sixteenth weeks, mental development may be severely affected in the perinatal and infantile periods by teratogens such as cytomegalovirus, ethyl alcohol and high doses of ionizing radiation.

Specificity is an important attribute of teratogenesis because nearly all known teratogens cause characteristic multiple congenital abnormalities (syndromes). Many other conditions or agents may have non-teratogenic but very serious or fatal effects on the foetus.

RADIATION EFFECTS

A series of biological events (a "response chain") must occur to reach a specific end-point of a developmental disturbance. The duration and type of response chain are affected by the magnitude of the dose (OE88) but are primarily dependent on the developmental stage at the time of irradiation.

Prenatal and neonatal deaths

Evidence for prenatal and neonatal death in human populations caused by irradiation of the pregnant mother and embryo is negligible. As a result, the results of animal experiments have been extrapolated to human populations. Problems associated with the extrapolation of animal data to predict the human experience are discussed later in this section.

The incidence of radiation-induced prenatal death is highest after irradiation in the early stages of development, particularly during the preimplantation period - that is, before the embryo becomes attached to the uterine wall. In general, irradiation at this stage either causes death or results in a normal individual. Despite the radiosensitivity of individual cells, the conceptus during the preimplantation period is characterized by a high capacity for regeneration and structural reorganization after radiation damage. Studies in rodents exposed during blastogenesis provide no indications of persistent effects below 0.1 Gy (OE88). After implantation, the embryo and foetus are less sensitive to radiation-induced death, the sensitivity approaching adult values at later stages of foetal development.

It is suggested (NR88) that human LD/50 values for acute exposure at gestational ages 1 week and 5-7 weeks be set at 1.0 and 1.5 Gy respectively. For 21 weeks to term the dose-response relationship for lethality is taken to be the same as adults (i.e., an LD/50 of 3.0 Gy).

Radiation-induced Central Nervous System Disorders

There are a number of ways that ionizing radiation can interfere with CNS development. Radiation effects can arise because of:

(a) the death at mitosis of neuronal or glial precursors or both, or the killing of postmitotic (but still immature) neurons;
(b) an alteration of normal cell migration pattern, due to an effect on the cell surface phenomena necessary for movement of neurons, or because of the premature death of glial cells that serve to guide the migrating neurons;
(c) impaired development of neuronal connections (synaptogenesis); or
(d) an alteration in the timing of programmed cell death during the development of the brain or its adnexa (visual, auditory, olfactory neurosensory structures and their neural pathways for interaction with the cerebral cortex).

Developmental age is the most important single factor in determining the nature of the insult to the embryo or foetus resulting from exposure to ionizing radiation.

Table 1

Temporal pattern of brain development and possible injury from radiation or other agents
(taken from UN90)

Gestational age (weeks)	Developmental Features	Possible Damage
3 - 4	Formation of neural tube	Defective fusion of the neural tube (dysraphic abnormalities)
4 - 10	Formation and fusion of cerebellar plates	Cerebellar agenesis or hypoplasia
3 - 12	Early multiplication of cells in ventricular zone	Small head size
6 - 16	Neuronal migration	Heterotropic grey matter
8 - 15	Cortical histogenesis	Mental retardation
24 - 33	Thalamocortical innervation	Abnormal cortical differentiation
24 - 38	Involution of subpial granual layer	Marginal heteroptias

Human Incidence of Radiation-Induced CNS Effects

Few population-based studies on the central nervous system effects of prenatal radiation exposure exist. Present knowledge rests mainly on observations of those exposed in utero at Hiroshima and Nagasaki, and to a lesser degree on studies carried out on children who were prenatally exposed to radium or X-rays in the course of radiotherapeutic treatment of their mothers, and on comparative embryological studies. Among these, however, the size, length of study, variability in dose and developmental age at exposure make the experience of the Japanese atomic bomb prenatally exposed survivors the most important source of data.

A study of about 1600 children exposed in utero at Hiroshima and Nagasaki to various acute radiation doses and at various developmental stages has shown about 30 cases of clinically severe mental retardation, mostly combined with micro-encephaly, with a greater incidence than expected in the higher dose groups. For comparison purposes, it is useful to note that the normal background frequency of severe mental retardation was 8 per 1,000 or 0.8%.

So far only two conspicuous effects on brain growth and development have emerged in this group, some being cases of severe mental retardation and some of small head size without apparent mental retardation. Additionally, groups within the survivors have shown an increased frequency of unprovoked seizures, significantly reduced intelligence scores and performance in school, and reduced performance on two neuromuscular tests-grip and fine motor coordination.

Severe Mental Retardation

The least vulnerable period is 0-7 weeks after fertilization. Irradiation during this period has not caused any radiation-related cases of severe mental retardation. However, it should be noted that within this interval a higher proportion of embryos fail to survive gestation, and it may be that brain damaged embryos are less likely to survive to an age where their handicap can be recognized clinically.

Mental retardation occurred most commonly from irradiation between 8 and 15 weeks of gestation and to a lesser degree between 16 and 25 weeks. No effect was observed following exposures later than 25 weeks. In foetuses exposed to radiation in the period 8-15 weeks after fertilization, the prevalence of severe mental retardation at estimated mean doses to the mother's uterus of 0, 0.05. 0.23, 0.64 and 1.38 Gy was respectively 0.8%, 4.5%, 3.4%, 25%, and 75%. The period of maximum sensitivity (8-15 weeks) corresponds with the timing of both of the major waves of neuronal proliferation and migration within the cerebral cortex. Within this exceptionally vulnerable period, damage expressed as the frequency of subsequent severe mental retardation is directly related to the dose received by the foetal tissues. Some 45% of foetuses exposed to 1 Gy (low-LET) at high dose rate in this period will be mentally retarded; this is a risk more than 50 times greater than that in the unexposed comparison group. There is a strong probability of a threshold of about 0.2 Gy (IC90).

In foetuses exposed to radiation in the period 16-25 weeks after fertilization, the prevalence of severe mental retardation at estimated mean doses to the mother's uterus of 0, 0.05, 0.23, 0.64 and 1.38 Gy was respectively 0.6%, 1.8%, 0%, 0%, and 37.5% (Figure 1). Damage to the foetus 16-25 weeks after fertilization seems linear-quadratically or quadratically related under the revised (DS86) dosimetry, with a threshold in the neighbourhood of 0.6-0.7 Gy. The risk at 1.0 Gy is about a quarter of that following exposure in the most sensitive 8-15 week period.

Reduced Intelligence Test Scores

Mental impairment of lesser severity is also apparent in children exposed in utero. Schull et al. (Sc88) have reported intelligence test scores at 10-11 years of age for individuals exposed prenatally to the A-bombings of Hiroshima and Nagasaki. Using the DS86 dosimetry, no evidence of a radiation-related effect on intelligence was obtained for individuals exposed 0-7 weeks after fertilization or in the 26th or subsequent weeks after fertilization. For individuals exposed at 8-15 weeks after fertilization (and, to a lesser extent, for those exposed at 16-25 weeks), the mean tests scores suggest a progressive linear downwards shift in I.Q. scores with increasing exposure. Those exposed to 1 Gy during the most sensitive period of brain development (between 8 and 15 weeks) exhibit a loss in intelligence of 25-30 points, as measured by I.Q. testing. A similar but smaller shift is detectable in those irradiated in the period from 16 to 25 weeks (UN90, BE90, IC86). In terms of school performance, prenatal exposure to 1 Gy appears to imply a decrement in average school performance which is equivalent to the shift of an average individual from the middle 50 percentile to the lower 10 percentile. The decrease in intelligence test scores can be described as a general shift of the Gaussian I.Q. curve towards lower values of I.Q. (see Figure 2).

Because of the broad spectrum of intelligence levels in the population, it is postulated that the dose required to cause an I.Q. change large enough to make an otherwise normal individual mentally retarded would be high, while the dose that would shift an individual with potentially low I.Q. over the borderline into clinical mental retardation may be quite small (the magnitude of the required dose follows from the shift of 30 I.Q. points per Sv) (Figure 2) (IC90). No data on effects of radiation at low dose rate are available. The direct use of coefficients derived from the study of the Hiroshima-Nagasaki children probably overestimates the risk of radiation at low dose rate (IC91). The ICRP has assumed that the downward shift in IQ is proportional to dose, but also "the effect is presumed to be deterministic with a threshold related to the minimum shift in IQ that can be recognized" (IC91).

Prenatal Radiation Exposure and Carcinogenesis-Data from Human Medical Exposure

A number of human epidemiologic studies suggest an association between the risk of childhood cancer (within 15 years of birth), and prenatal exposure to diagnostic X-rays in utero. One of the most important is the Oxford Survey of Childhood Cancers (OSCC) which is an on-going case-control study (started in 1955) of childhood (under age 16) cancer deaths in Great Britain. Another notable investigation is that of McMahon, who originally surveyed 734,243 New England children, and later extended the program to include 1.43 million children born between 1940 and 1960. These and other case control studies show the relative cancer risk associated with prenatal X-rays to be greater than unity, taking into account the different populations studied and variations in project design and analysis methodology used by different investigators.

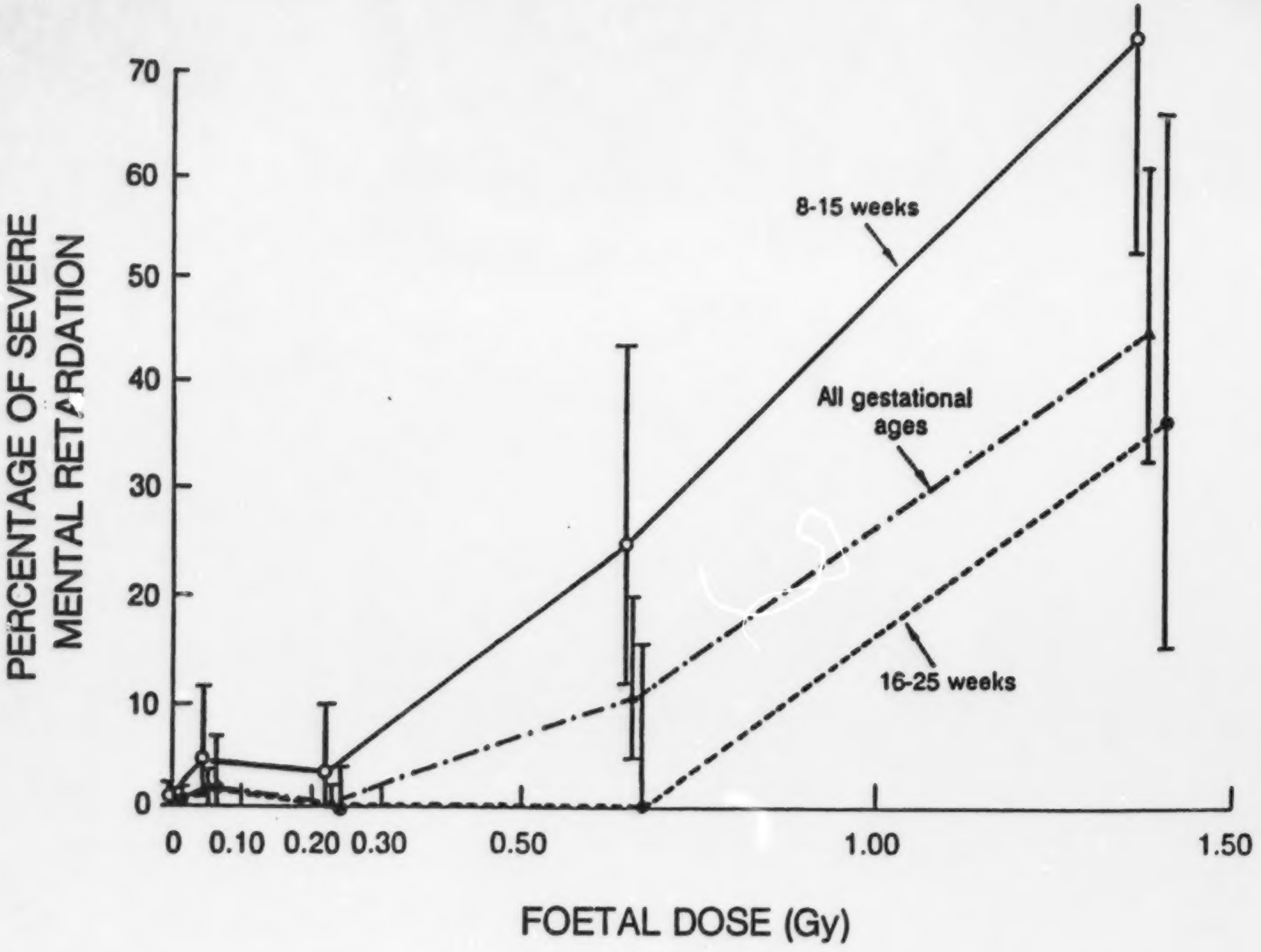

Figure 1. The percent age of severe mental retardation among those exposed in utero by dose and gestational age in Hiroshima and Nagasaki. The vertical lines indicate 90% confidence intervals.

From Beir V

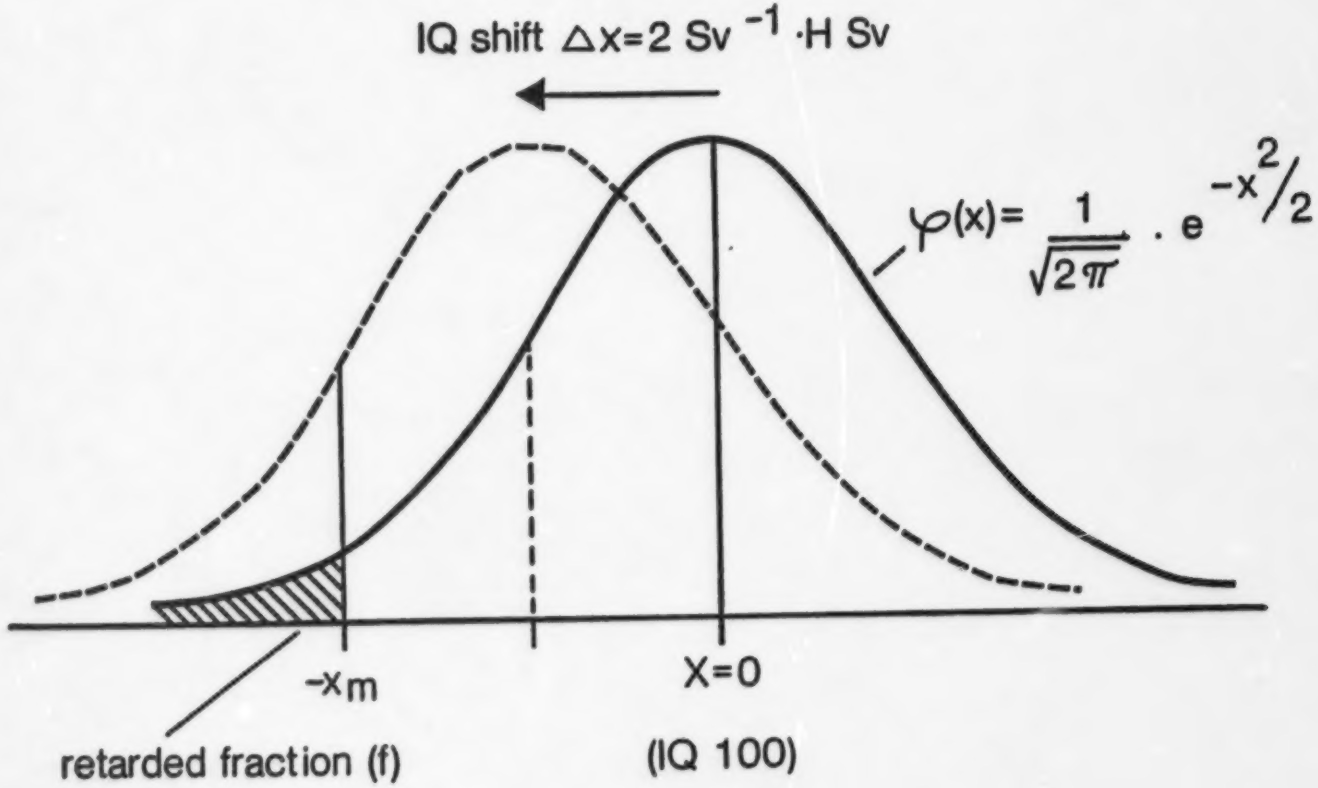

Figure 2. The shift of the IQ curve by 30 IQ units or 2 σ per Sv, i.e. $\Delta x = 2H$ if H is the dose equivalent expressed in Sv. The variable x is the number of standard deviations below (–) or above (+) IQ 100. x_m denotes the number of standard deviations below IQ 100 to classify an individual as mentally retarded.

From: Draft ICRP Recommendations (1990)

As the average radiation dose (BE90) to the foetus from a diagnostic radiography procedure is relatively low (in the order of 5-50 mGy), these data imply that susceptibility to radiation carcinogenesis is relatively high during prenatal life. Even after allowing for confounding factors, the weighted average (NR88) of the relative risks is 1.39 ± 0.08 (95% confidence limits).

NRPB (NR88) estimates the number of excess cancer cases following irradiation in utero to be 6×10^{-2} Gy^{-1} (low-LET), comprising 2.5×10^{-2} Gy^{-1} for leukemias and 3.5×10^{-2} Gy^{-1} for solid cancers. The assumption is that half of all childhood cancers are fatal, so therefore the number of excess cancer deaths is taken to be 3×10^{-2} Gy^{-1} (low-LET), comprised of 1.25×10^{-2} Gy^{-1} for leukemias and 1.75×10^{-2} Gy^{-1} for solid cancers. These estimates are derived from follow-up studies on children irradiated in utero by diagnostic X-rays with radiation doses up to a maximum of 10-20 mGy (low-LET), and are applicable for estimating risks at low doses and dose rates. A somewhat lower cancer risk value of 2×10^{-2} Sv^{-1} is suggested by UNSCEAR (UN77, UN86).

Mole (Mo90) compared the Adrian Commission foetal bone marrow dose estimates from diagnostic prenatal radiological procedures during 1958 with the cancer incidence in children born in the years 1958-61. The resultant risk coefficient for childhood cancer caused by X-ray examination in late pregnancy is $4\text{-}5 \times 10^{-2}$ Gy^{-1} with 95% confidence limits $0.8\text{-}9.5 \times 10^{-2}$ Gy^{-1}. This value applies equally to cancer incidence and mortality because the effect of therapy on survival with cancer was negligible during that period.

Japanese children irradiated in utero during the Hiroshima and Nagasaki bombings received much higher radiation doses (estimated up to 5 Gy) than the medically exposed populations. A study of cancer mortality up to age 10 in this group of 1250 children did not show an excess in childhood cancer, which is inconsistent with the findings from other studies of low dose medical prenatal exposure. However, a 40 year follow up study of 1630 Japanese survivors irradiated in utero show some indications that the risk may appear after the first 15 years of life, although the magnitude of the risk is uncertain.

UNSCEAR (UN88) offers some cautions about the use of risk factors for carcinogenesis arising from in utero irradiation.

"The lack of an effect on early childhood cancers even after higher doses in Japan is a ground for caution in interpreting the positive effects from medical irradiation studies, especially in the light of many possible confounding or biasing factors. In a similar way, the fact that early post-natal irradiation in Japan showed carcinogenic effects only many years later (agreeing with other data on post-natal radiation effects), whereas the medical series showed their effects soon after birth, raises serious radiobiological problems if both observations are strictly the result of radiation. The doubts are enhanced by the absence of serious effects in animal experimental data. ... However, it would be prudent to assume that prenatal irradiation does have an effect, especially with regard to leukaemogenesis."

Prenatal Exposure and Carcinogenesis - Animal Models

A number of factors make the extrapolation of the results of animal experiments to radiation-induced tumourigenesis in man uncertain. Discernible increases in the incidence of cancer and leukemia have not been demonstrated after exposures during blastogenesis or early organogenesis. This contrasts with an increasing susceptibility during the subsequent stages, including the prenatal period. Tumor-promoting or tumourigenic doses in animals have not been demonstrated below 0.1 Gy, and in most cases are higher than 0.5 Gy. After external exposure, brain, liver, lung, ovaries and mammary glands were the most common sites of tumours. Susceptibility to lymphoma induction is most evident after exposure during the advanced intrauterine period. Experimentally determined tumour induction by internal exposure to specific radionuclides is prominent during growth or onset of function in target organ. Striking examples of this are thyroid or bone tumours arising from intake of radioiodine or radiostrontium respectively, although many other examples are available.

INTERSPECIES RADIOSENSITIVITY AND TYPE OF RESPONSE

There are considerable difficulties in directly extrapolating in utero animal radiosensitivity data to humans. For example, independent of the relative rate of organogenesis, rodent embryos exhibit a high degree of differentiation as well as a more rapid cell proliferation in a much shorter time that in humans. In addition, because of the greater rapidity of rodent development, compensatory responses are restricted to much shorter periods than in humans; and these temporal shifts may result in an enhanced radiosensitivity during certain stages of development in the rodent. On the other hand, the special conditions that apply during human corticogenesis (greater migration of the neuro-blasts) may lead to similar or increased sensitivity of the human central nervous system.

Regardless of the different transition times, the sequence of qualitatively similar differentiation steps is identical in mammals and humans. Therefore radiation dependent, embryotoxic responses are parallel in various species when noxious agents act on homologous stages of development (see Figure 3). Accordingly, model experiments with laboratory mammals are of importance for detection and analysis of types of damage that are diagnostically difficult or impossible to determine in humans (OE88).

SUMMARY

Dose-effect relationships are given for prenatal and neonatal deaths, severe mental retardation, and cancer induction following prenatal irradiation.

1. Mortality

Mortality depends upon the period of gestation at which exposure occurs. LD/50s of 1 Gy, 1.5 Gy and 3 Gy are likely following exposure at 1 week, 5-7 weeks and 21 weeks to term (NR88). There is evidence to suggest lethal effects in the embryo with a threshold of 0.1 Gy or less (IC90).

2. Malformations

During the period of major organogenesis, malformations may be caused in organs under development of the time of exposure. These effects are deterministic in character with a suggested threshold in humans of about 0.05 Gy.

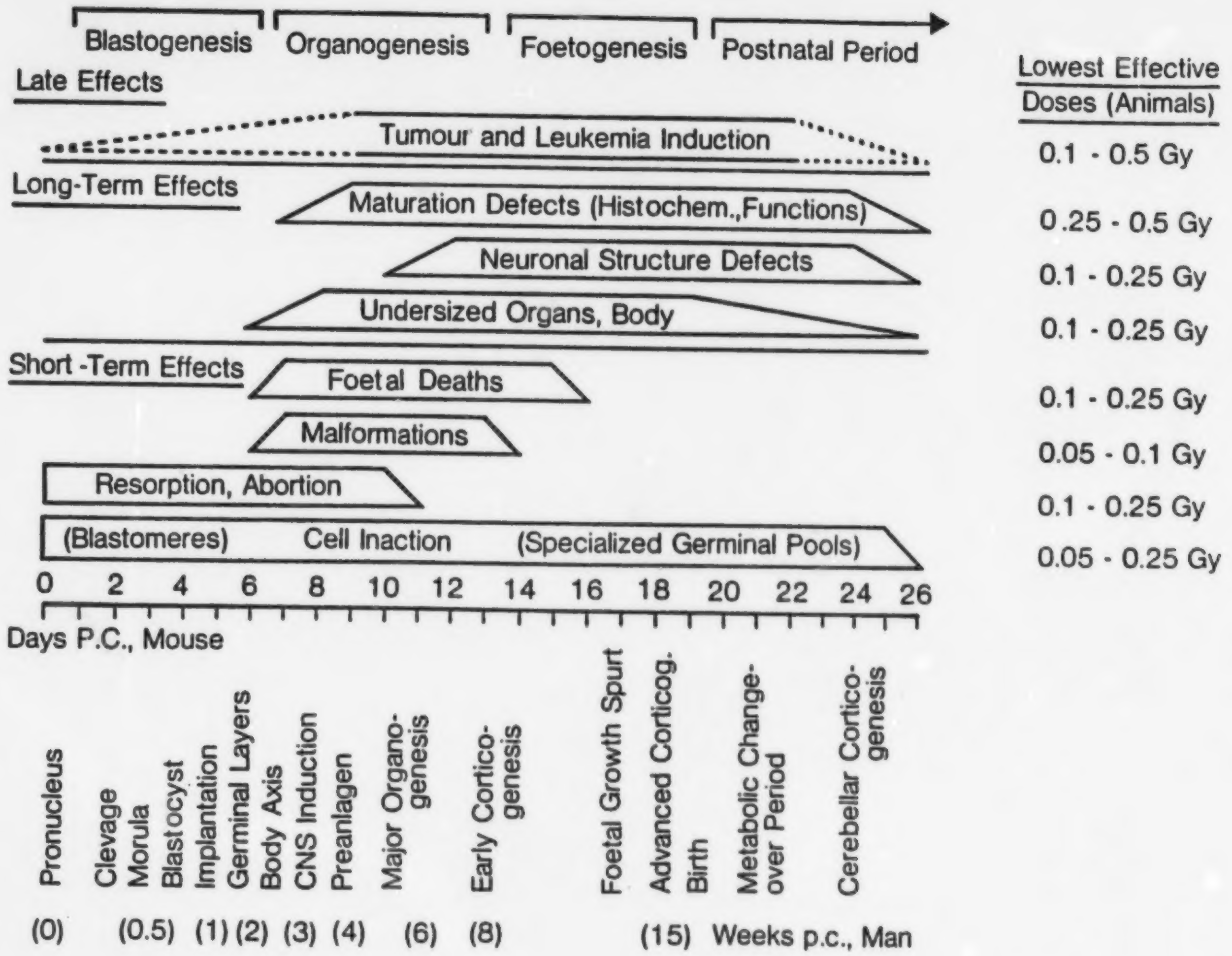

Figure 3

Homologous stages of mouse and human development and induction periods for short-term, long-term and late effects of ionizing irradiation. Time scale for the major developmental periods (at the top) as well as lowest effective dose (on the right) refer particularly to mice.

3. Severe mental retardation

The developing human brain has been shown to be highly vulnerable to radiation damage between 8 and 15 weeks of gestation. The risk of severe mental retardation resulting from exposure during this period is determined to be about 40 x 10^{-2} Gy^{-1} (low-LET) with a possible threshold of a few hundred milligray. This risk is based on the Japanese population exposed at high dose rates. No dose rate effectiveness factor (DREF)[1] has been applied in the absence of any comparative data at low and high dose rates.

4. Reduced intelligence

For individuals exposed at 8-15 weeks after fertilization, and to a lesser extent those exposed at 16-25 weeks, the mean test scores suggest a progressive linear shift downwards in I.Q. scores with increasing exposure. Those exposed to 1 Gy during the most sensitive period of brain development between 8 and 15 weeks exhibit a loss in intelligence as measured by I.Q. testing of about 30 points. A similar but smaller shift is detectable in those irradiated in the period from 16 to 25 weeks. No evidence of a radiation-related effect on intelligence among those individuals exposed within 0-7 weeks after fertilization or in the 26th or subsequent weeks was obtained.

5. Childhood cancers

Irradiated foetuses seem to be susceptible to childhood leukemias and other childhood cancers which are usually expressed before age 10. Estimates of the cancer risk have been obtained by combining the excess risk observed with estimate of dose to the foetus due to obstetric X-rays. This yields a risk of 2.5 x 10^{-2} Gy^{-1} for leukemia and of 3.5 x 10^{-2} Gy^{-1} for other cancers; half of these cases are expected to be fatal. These risk coefficients are applicable to high and low dose rates and over the entire period of gestation.

There is disagreement between the data from the Japanese bombing survivors prenatally exposed to large doses and data from large populations of children who received small doses of prenatal radiation for medical reasons. The estimated risk of childhood cancers and leukemias up to age 10 years ranges from 2 x 10^{-2} Sv^{-1} (UN77, UN86) to 6 x 10^{-2} Sv^{-1} (NR88) taking these differences into account. Constancy of the risk throughout pregnancy is assumed in the absence of convincing data to the contrary. The most recent report from the ICRP suggests that the lifetime risk of fatal cancer or "nominal fatality probability coefficient is, at most, a few times that for the population as a whole" (IC91).

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[1] DREF: a factor used to derive risk estimates of exposure to low-LET radiation at low dose rates, from risk estimates calculated from data associated with exposures at high dose rates.

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ANNEX G

Genetic Risk Estimates

(D.K. Myers)

There is substantial evidence that radiation-induced changes in lower organisms are truly stochastic (i.e. incidence directly proportional to accumulated dose without any threshold dose), but no reliable evidence exists for the induction of an excess of genetic disorders in the children of humans exposed to high radiation doses. For example, there is no statistically significant increase in potential genetic disorders among the children born to parents of whom at least one was a Hiroshima-Nagasaki survivor. Some 70 000 pregnancies were studied although less than a third of these involved a parent who received 0.01 Gy or more in 1945 (Sc81, BE90). There are non-significant trends towards a possible increase in potential genetic effects at higher doses. When all of the relevant data are consolidated, this trend suggests that the dose of gamma radiation required to double the natural mutation rate might be in the region of 2 Sv (Ne90). The 95% confidence limits on these doubling doses cover a range from about 0.2 Sv to infinity (Sc81, BE90). The untoward pregnancy outcomes analyzed include major congenital defects, stillbirths and deaths during the first week of life, all of which are expected to increase following an increase in deleterious mutations. Other endpoints examined were survival of live-born children to age 17, chromosomal abnormalities and electrophoretic variants in blood proteins.

Because none of these effects is statistically significant, scientific committees responsible for quantitative assessment of the genetic risks of radiation have relied primarily on other methods of estimating these risks. The basic requirements include a measure of the normal incidence of genetic disorders in human populations, the contribution of new mutations in each generation to these incidences, and the rate of induction of new mutations per unit dose of radiation.

There are many different diseases and categories of diseases which have a strong genetic component. For simplicity, only three general categories are considered here: (a) monogenic, which includes dominant, recessive and X-linked mutations of a single gene, (b) chromosomal, which are due to changes in the structure or the number of chromosomes in the germ cell, and (c) multigenic (multifactorial), where the disease is associated with the interaction of a considerable number of genes and of the environment. Most congenital abnormalities, i.e. defects which are evident at or shortly after birth, are normally classified as multigenic; congenital abnormalities were, in fact, responsible for about half of the total number of disorders that were recognized as multigenic (multifactorial) prior to 1986 by the BEIR and UNSCEAR Committees.

Table 1 summarizes the normal incidence of genetic diseases in industrialized countries as estimated in recent reviews by scientific committees. A major change occurred in 1986 with an increase in normal incidence of multigenic (multifactorial) diseases from 9 to 66 per 100 live births. This increase was due to inclusion in this category of a variety of well-known multigenic disorders such as type II (adult onset) diabetes, schizophrenic and affective psychoses, glaucoma, essential hypertension, myocardial infarction, varicose veins, asthma, gastro-intestinal ulcers, and rheumatoid arthritis, which normally appear in adulthood and for which there is good evidence for a heritable component. This trend has been continued by the most recent BEIR Committee; since many persons may suffer from more than one of these disorders, the actual number of persons affected will be somewhat lower than the incidence of all potential diseases.

Table 1

Normal incidence of genetic disorders or partially genetic disorders in human populations

Category	Incidence per 100 live births				
	UNSCEAR 1977	BEIR 1980	UNSCEAR 1982	UNSCEAR 1986, 1988	BEIR 1990
Monogenic	1.1	1.1	1.3	1.3	1.3
Chromosomal	0.4	0.6	0.34	0.4	0.4
Multigenic (multi-factorial)	9.	9.	9.	66.	120.
Total	11.	11.	11.	68.	122.

Another major development since 1977 has been consideration of the potential impact of genetic disorders in terms of years of life lost or seriously impaired. Data on years of life lost or seriously impaired due to the normal incidence of selected categories of genetic disorders are summarized in Table 2 for a society in which human life expectancy at birth is about 70 years. For those categories examined, about 18 years of life on average are lost or seriously impaired per case of genetic disorder in the normal population. This value is similar to that for years of life lost per radiation-induced fatal cancer.

A variety of methods have been employed in order to estimate radiation-induced mutation rates and their impact on human populations. Some of the basic data include incidence of coat-colour mutations in progeny after exposure of male and female mice to various doses of radiation at various dose rates, incidence of skeletal abnormalities and of cataracts in progeny after irradiation of male mice, and chromosomal translocations in a wide variety of species. Using various assumptions to extrapolate from the experimental data to impact in terms of human diseases, the risk estimates given in Table 3 were derived. The cumulative total of affected progeny over all generations after a single exposure (Table 3) is equal to the increase in equilibrium value after continuous exposure of the parents in each generation.

Table 2

Years of life lost or seriously impaired due to genetic or partially genetic disorders (UNSCEAR 1986)

Category	Per 100 live births		
	Incidence	Total years life lost	Years of life seriously impaired
Monogenic	1.3	19	15
Chromosomal	0.4	9	9
Congenital abnormalities	5.9	45	39
Total	7.6	73	263

Table 3

Estimates of radiation-induced genetic or partially-genetic disorders in human populations exposed to 100 person-Sv prior to conception of children

Category	UNSCEAR 1977	BEIR 1980	UNSCEAR 1982	UNSCEAR 1986, 1988	BEIR 1990
Cases per live birth in first generation:					
Monogenic	0.2	0.05-0.65	0.05-0.15	0.05-0.15	0.06-0.35
Chromosomal	0.4	<0.1	0.003-0.065	0.005-0.10	<0.06
Cumulative total over all generations:					
Monogenic	1.	0.4-2.	1.	1.2	1.
Chromosomal	0.4	slight	0.04	0.04	<0.06
Multigenic (multifactorial)	0.45	0.2-9.	0.45	(a)	≥0.1-1
Total	1.9	0.6-11.	1.5	≥1.2	≥1.1-2

(a) No estimate given.

The genetic risk estimates given in the 1977 UNSCEAR report are similar to those recommended for radiation protection purposes in ICRP Publication 26. The 1980 BEIR report provided a range of values extending to much higher estimates than suggested by the 1977 and 1982 UNSCEAR reports (Table 3). This difference was due in part to the use of a range of doubling doses

(0.5-2.5 Sv) for mutation rates rather than the single value (1 Sv) adopted in the UNSCEAR reports; more importantly, the 1980 BEIR Committee assumed that new mutations might be responsible for 5 to 50% of the incidence of multigenic diseases while the UNSCEAR Committees assumed a single value of 5% for the mutational component. This topic obviously poses problems for radiation geneticists because of the paucity of decisive information. However, the 1990 BEIR report has adopted a position which is rather similar to that of recent UNSCEAR reports.

The recent increase in the assumed incidence of multigenic disorders in the normal population (Table 1) also raises some problems. Most of this increase relates to diseases which appear in older persons; thus, the impact in terms of years of life lost or seriously impaired should be fairly small. Moreover, this increase should not in itself have any appreciable influence on the percentage of all genetic disorders which could be attributed to a given radiation exposure. On the basis of the risk estimates and the normal incidences given in the 1977 UNSCEAR report (Tables 1 and 3), background exposures of 2 mSv per year for 30 years to both parents (total = 60 mSv) would be responsible for about 1% of the normal incidence of genetic and partially genetic diseases. When these percentages are recalculated in the same manner but using a 10-fold increase in normal incidence of multigenic diseases (Table 1), there is little change in percent of normal incidence that could be attributed to background exposures of 1 mSv per year, even though the absolute numbers may increase appreciably. The importance of multigenic traits is emphasized by the consideration that these inherited traits include longevity as well as most of the other characteristics of a species.

The 1977 ICRP recommendations assumed that the total probability of radiation-induced genetic disorders in the children and grandchildren of exposed persons was 1×10^{-2} Sv^{-1} received prior to conception. Since the mean age of parents at reproduction is about 30 years, the genetic risk averaged over a complete population with a life expectancy of 70 years would be 0.4×10^{-2} Sv^{-1}.

The 1990 draft of the proposed new ICRP recommendations suggests that radiation-induced genetic disorders should be summed over all subsequent generations rather than over the first two generations only. Using some reasonable assumptions, the probability of all genetic diseases summed over the first two generations only was calculated to be about 0.4×10^{-2} Sv^{-1} averaged over the whole population. It was assumed that severe multifactorial disorders would be eliminated within the first two generations, and that the total risk of serious genetic disorders summed over all subsequent generations would be about 1×10^{-2} Sv^{-1} averaged over a complete population with a life expectancy of 70 years. Since a working population is exposed to occupational sources of radiation only from age 18 on, the total genetic risk should be somewhat smaller and is taken to be about 0.6×10^{-2} Sv^{-1} for occupational exposures. This risk estimate applies to serious genetic disorders only and represents a summation over all subsequent generations of descendants.

A recent study (Ga90) has suggested a possible association between occupational exposures of fathers and development of childhood leukemia in their offspring. If confirmed, this association might require an appreciable increase in genetic risk estimates. There is no increase in cancer before age 20 in the children of parents exposed to radiation at Hiroshima and Nagasaki (Yo90). The ACRP will keep this situation under review and issue any appropriate statement at a later date when more data are available.

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ANNEX H

Epidemiological Studies of Workers
(D.K. Myers)

Estimates of the probability of developing cancer after exposure to low doses of radiation at low dose rate are based on data derived from the study of groups of persons, notably the Hiroshima-Nagasaki survivors, who were in the past exposed to relatively high radiation doses at high dose rate. There are many uncertain-ties involved in this extrapolation. These uncertainties would disappear if it were possible to derive risk estimates directly from the study of workers or other groups exposed to small increases in low-LET radiation at low dose rate.

Epidemiological studies of radiation workers usually show lower rates of cancer mortality than observed for persons of the same age and sex in the general population (Fig. 1). In general, the differences are not statistically significant and the observed decreases are attributable to healthy worker effects. It is, however, of some interest that workers exposed to high doses, e.g., an average of 420 mSv recorded occupational dose over an average of 24 years of employment at Chalk River (We86), also tend to show low rates of cancer mortality (Fig. 1).

Table 1 summarizes the characteristics of the major studies of persons exposed to measured increases in low-LET radiation at work and compares these groups with the Hiroshima-Nagasaki survivors. In general, the cumulative occupational exposures of the workers are considerably lower than those of the Japanese bomb survivors, the number of workers exposed to doses above 100 mSv is also relatively small, and the total number of cancer deaths to date is smaller than the total in the exposed group of bomb survivors. The precision of risk estimates derived from studies of radiation workers is thus considerably lower than those which can be derived from the study of the Hiroshima-Nagasaki survivors. Estimates of the probability of radiation-induced cancer have been given for three of the studies of workers listed in Table 1. In one case the risk estimates are similar to those for the bomb survivors but the confidence limits on the estimates are much broader. In the other two cases, the risk estimates were negative; that is to say, there was a trend towards fewer cancers with increasing radiation exposures..

Trends in relative risk of fatal cancer in the U.S. radiation workers are compared with those of the bomb survivors in Table 1. The upper 90% confidence limit on the trend for U.S. radiation workers is essentially identical with the best estimate for the Hiroshima-Nagasaki survivors (using DS86 values), while the lower confidence limit is strongly negative. It should be noted that the data for U.S. radiation workers refer to adult males only while those for the bomb survivors refer to a complete population of all ages and both sexes; moreover, no dose-rate effectiveness factor (DREF)[1] has been applied to the Hiroshima-Nagasaki data given in Table 1.

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[1] DREF: a factor used to derive risk estimates of exposure to low-LET radiation at low dose rates, from risk estimates calculated from data associated with exposures at high dose rates.

Table 1
Characteristics of some studies of workers exposed to low-LET radiation and of Japanese bomb survivors

	AECL (Ho87)	AWE (Be88)	UKAEA (Be85)	Sellafield (Sm86)	Hanford - Oak Ridge - Rocky Flats (Gi89)	Hiroshima - Nagasaki (Sh87, Sh88, Sh90)
Number of persons						
- exposed	6,626	9,389	20,382	10,484	35,933	41,719
- not exposed	(c.3,000)	13,163	19,164	3,843	-	34,272
Number of persons with >100 mSv	-	140	1,675	3,843	2,910	18,398
Average exposure (mSv) of irradiated group	47	8	32	124	32	about 300
Average length of follow-up (years)	16.7	18.5	16	21.6	20	29
Number of cancers in irradiated group	150	275	393	396	1,036	3,435
% female	0	9	0	19	0	60
SMR compared to general population(a)						
- leukemia	0.46	0.44	1.10	0.82	0.92	-
- all cancers	0.90	0.79	0.75	0.95	0.79	-
SMR compared to non-exposed group						
- leukemia	0.38	0.42	0.96	(b)	-	2.10
- all cancers	1.06	1.03	0.89	1.00	-	1.14
Excess cancers(c) per 10^6 person-years per 10 mSv						
- leukemia	-	-	+ 2.2 (-3 to +12)	-	negative (negative to +2.4)	+2.9(+2.4 to+3.5)
- all cancers	-	-	+12.5 (-22 to +52)	negative (-22 to +13)	-30 (negative to +11)	+10.1 (+8.0 to +12.4)

(a) SMR is standard mortality ratio, which is equivalent to relative risk.

(b) No meaningful value given since there was only 1 leukemia death in the non-exposed group.

(c) Risk estimates based on a linear fit to trends within the exposed group; values in brackets represent the 90% confidence limits.

The BEIR V Committee (BE90), which did not have the latter data (Gi89b) available for review, concluded that the studies of radiation workers "have provided no evidence to date that risk estimates for leukemias and other types of cancer combined are in error, based on extrapolation from high dose studies". Gilbert and co-workers (Gi89b) indicated "that estimates obtained through extrapolation from high-dose data do not seriously underestimate risks of low-dose exposure, but leave open the possibility that extrapolation may overestimate risks". Potential conflicts between combined data on radiation workers and data for the bomb survivors would be minimized by the suggested application (BE90, UN88) of an appreciable DREF to the Hiroshima-Nagasaki data.

Complete populations living in areas of unusually high exposure to external gamma radiation from natural sources have also been studied. There does seem to be an inverse correlation between cancer mortality and natural radiation levels in the U.S.A. (Fr75), which might be due to other confounding factors such as altitude or lifestyle. Extensive observations have also been carried out in high background areas of China (Lu85), where the majority of the population have lived in the same area for several generations. The increase in external gamma radiation in the high background areas amounts to 2-3 mSv per year over that in adjacent control areas. The rate of cancer mortality, as well as of hereditary diseases and congenital defects, was somewhat lower in the high background areas although the differences were not statistically significant (Lu85).

Some authors have interpreted the above data as evidence in favour of possible beneficial effects of low levels of radiation. This would mean that attempts to reduce low levels of exposure even further might in fact result in the reduction of potential health benefits.

Arguments against this interpretation are the observation of increased incidence of chromosomal abnormalities in white blood cells of persons living in high background areas (despite the non-significant decrease in cancer mortality) (BE90, Lu85) and the probability that any additional damage to cellular DNA is likely to increase cancer development in human populations which already exhibit high cancer mortality under normal circumstances.

However, the studies on radiation workers and on whole populations exposed to low levels of low-LET radiation do suggest a need for caution. It is generally considered reasonable and prudent to assume a linear non-threshold dose-response relationship for the probability of radiation-induced cancer; this assumption is unproven at low doses and low dose rates and is, in fact, unprovable for very small increments in dose. Low levels of radiation are not a major factor affecting cancer mortality; other factors play a more important role. Sufficient information on workers exposed to low-LET radiation is available to indicate that risk estimates obtained through extrapolation from high dose data do not seriously underestimate risks of low-dose exposure, but leave open the possibility that this extrapolation may overestimate risks. The data offer support for the existence of a dose rate effectiveness factor (DREF). The magnitude of this DREF cannot be quantified from the present data on radiation workers. Because there is no proof that low levels of radiation do cause cancer, it would be prudent to speak of potential health detriments of exposure to low levels of radiation. Continued international collaboration on the epidemiological studies of workers is essential in order to minimize uncertainties concerning the carcinogenic effects of low doses of radiation at low dose rate.

The importance of continued followup and consolidation of data from different countries is emphasized by a recent report on Oak Ridge workers (Wi91). Addition of another seven years of followup to earlier data resulted in a statistically significant increase in cancer mortality among these workers (Wi91). These results differ from those observed in the study of Chalk River workers, where cancer mortality during the last four years of followup was somewhat lower than that observed in previous years (We90). Caution is obviously required to avoid over-interpretation of the results from any one particular study of radiation workers

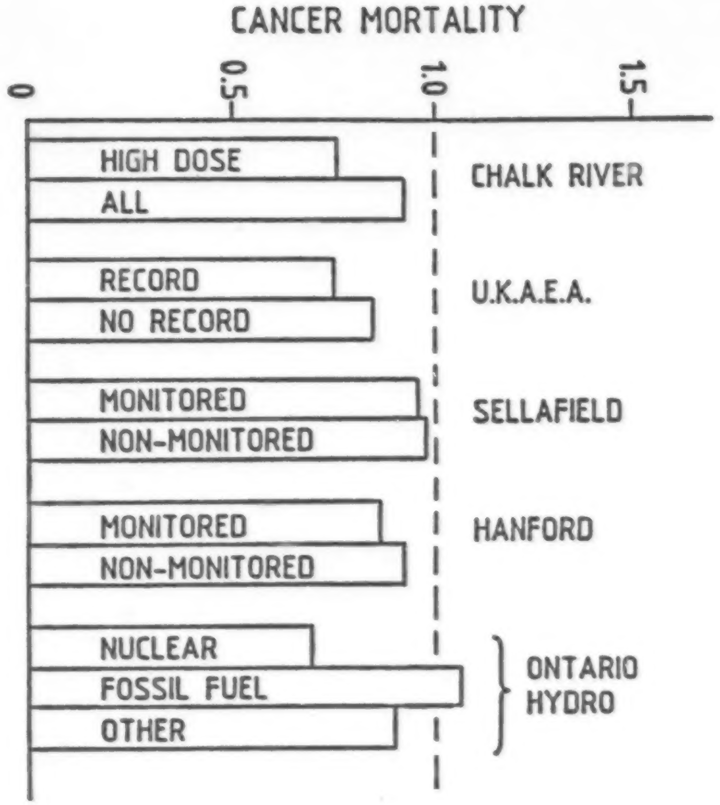

Figure 1. Standard mortality ratios for fatal cancers observed in various groups of workers. The horizontal dashed line indicates the expected value for persons in the general population of the same age and sex. The literature references for these five studies are An87, Be85, Gi89a, Sm86, We86.

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ANNEX I

Risks of Radon and Thoron Progeny

(D.K. Myers)

The health effects of inhalation of radon progeny have been reviewed many times, most recently by the Advisory Committee on Radiological Protection in a document titled "Radiological Hazards to Uranium Miners". The present annex is based in large part on the information in that document (AC90). The ICRP intends to publish a report on the same topic in the near future.

Studies of workers in a wide variety of uranium and other mines in various countries have consistently shown an excess of lung cancers which can be attributed to high concentrations of radon progeny in these mines. There are at present about 7 studies from which quantitative estimates of excess lung cancers per unit exposure to radon progeny can be derived. These risk estimates cover a seven to ten-fold range. A relative risk coefficient of 1% per WLM* and an attributable risk coefficient of 10^{-5} per WLM per year are reasonably representative of the derived risk estimates. Dosimetric calculations suggest that radiation doses, and hence potential hazard, to other tissues in the body should be very small compared to those in the respiratory tract.

The lung cancer data from the epidemiological studies of miners are compatible with a linear, non-threshold dose-response relationship, at least up to a cumulative exposure of 300 WLM. The minimum latent period to appearance of radiation-induced lung cancers is five years after first exposure. The data suggest that the maximum increase in lung cancers occurs 10-15 years after exposure, after which the incidence of radiation-induced lung cancers decreases towards zero by 20-30 years after exposure. The time course of the appearance of lung cancers in miners after exposure to radon progeny is thus different from that observed in the Hiroshima-Nagasaki survivors who were exposed to whole body gamma radiation in 1945 (UN88), where increased lung cancer risks appear to continue for at least 40 years after exposure. However, the miners were all adult men, of whom about 70% were cigarette smokers, while the Japanese bomb survivors were about 40% men and were of all ages. Cigarette smoking is known to increase the risk of lung cancer in miners exposed to radon progeny, at least during the earlier years of follow-up. It has been suggested that cigarette smoking promotes the early appearance of radiation-induced lung cancers in miners (UN82), with the result that most of the total lung cancers initiated by radiation have appeared within the first two decades after exposure to radon progeny. (A direct comparison with the effects of cigarette smoking on lung cancer incidence in the Japanese bomb survivors is complex. Although the average level of cigarette smoking was low at the time of exposure to radiation in 1945, levels have increased in recent years to about 84% of the males and 16% of the females (Sh88). In the groups exposed to the higher radiation doses, excess lung cancers in males appear to have largely disappeared by 35-40 years after exposure while the excess in females shows little sign of decreasing at 40 years after exposure (Sh88). Overall, the absolute risk of lung cancer does not differ between the sexes while the relative risk is lower for males, probably reflecting the higher background rates of lung cancer in males as a result of their higher cigarette consumption. The limited and incomplete data available to date do not contradict the hypothesis that cigarette smoking promotes the appearance of radiation-induced lung cancers.)

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* The WLM or working level month equals exposure to a potential alpha energy from the short-lived progeny of radon-222 of 0.0035 $J.h.m^{-3}$.

Most of the miners in these study groups are still alive. The limited period of observation makes it necessary to extrapolate well beyond the actual period of epidemiological follow-up in order to derive lifetime risk estimates. Various models have been used to do so. The results of these extrapolations in terms of lifetime risk estimates are summarized in Table 1. An average lifetime risk of 3×10^{-4} WLM^{-1} might be considered reasonable based on data for adult male miners.

This average risk per WLM is equivalent to about 18 mSv of whole body gamma radiation using the weighted risk coefficient recommended by the ICRP in 1977 (IC77), but would be equivalent to about 5 mSv using the aggregated detriment coefficients for workers suggested in the 1990 ICRP recommendations.

There are no epidemiological data from which risks from inhalation of thoron (radon-220) progeny can be derived. Dosimetric calculations suggest that the risk per WLM of thoron progeny is about one-third of that per WLM of radon progeny. Roughly 60-70% of the potential health hazard due to inhalation of thoron progeny is associated with irradiation of the lung, as it is in the case of radon progeny, while the other 30-40% is thought to be due to irradiation of other tissues.

There are many uncertainties in the extrapolation of these lifetime risk estimates for miners to a whole population of all ages and both sexes. Attempts to do this have resulted in values of 2×10^{-4} (IC87) to 3.5×10^{-4} (BE88) WLM^{-1}. A risk of 3×10^{-4} WLM^{-1} for the general population would mean that one WLM would be equivalent to about 4 mSv of whole body gamma radiation using the aggregated detriment that was suggested in the 1990 ICRP recommendations.

Table 1

Some estimates of the lifetime risk of lung cancer attributable to inhalation of radon progeny by adult male miners

Reference	Range of lifetime risk estimates based on epidemiological data ($\times 10^{-4}$ WLM^{-1})	Assumed Average risk ($\times 10^{-4}$ WLM^{-1})
ICRP-32 (IC81)	1.5 - 4.5	3
ACRP-1 (AC82)	1 - 6	3
BEIR IV (BE88)	-	5
UNSCEAR (UN88)	1.5 - 4.5	3
ICRP draft (IC90)	1 - 4	2.5
ACRP-12 (AC90)	1 - 6	3

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ANNEX J

ICRP 1990 Recommendations

(A.M. Marko, D.J. Gorman, and D.K. Myers)

I. INTRODUCTION

From a historical perspective the annual limit for occupational exposure of the whole body was reduced by a factor of about 3 between 1934 and 1950, and by a further factor of 3, to the equivalent of about 50 mSv, by 1965.

The intent of ICRP recommendations concerning procedures to control exposures to ionizing radiation is to ensure that radiation remains a minor component of the spectrum of risks to which we are all exposed. ICRP also believes that the degree of environmental control needed to protect individual humans to the degree currently thought desirable will ensure that the survival of other species is not put at risk. This topic has been considered previously (see, for example, Wh74, IA76, IA88, My89) and is currently under review by international agencies.

Non-stochastic effects are now called "deterministic". Deterministic effects are only manifested after a threshold dose is achieved. Stochastic effects (cancers and hereditary effects) are assumed to follow a dose-response relationship that has no threshold and is linear with accumulated dose at low dose rates.

II. DOSIMETRIC ASPECTS

The ICRP (IC90) has introduced a new term "effective dose" to replace the older term "effective dose equivalent" and "equivalent dose" to replace "dose equivalent". Effective dose is a doubly weighted absorbed dose, consisting of absorbed dose multiplied by W_R, or radiation weighting factors which depend on type and energy of radiation, and W_T, the tissue weighting factors which account for different sensitivities of tissues to radiation. Tables 1 and 2, taken from the ICRP 1990 Recommendations give numerical values for W_R and W_T.

The time integral of the equivalent dose rate over a specified period of time after intake in years is called the committed equivalent dose. The time period is usually 50 years for adults and 70 years for children. Also the committed effective dose is defined in a similar way. The ICRP states that when the terms committed equivalent dose or committed effective dose are used, it is implied that both the equivalent dose or effective dose resulting from external sources of exposure and the 50-year committed equivalent dose or committed effective dose from any intake occurring in that same time period, are included.

The dosimetric quantities referred to above all relate to the exposure of the individual. ICRP uses further quantities related to exposure of groups or populations. These quantities take account of the number of people exposed to a source by multiplying the average dose to the exposed group from the source by the number of individuals in the group. In this way collective equivalent dose and collective effective dose can be determined.

Table 1
Radiation Weighting Factors(1)
(Adapted from Table 1, IC91)

Type and energy range		Radiation weighting factor, W_R
Photons, all energies		1
Electrons and muons, all energies		1
Neutrons, energy	<10 keV	5
	>10keV to 100 keV	10
	>100 keV to 2 MeV	20
	>2 MeV to 20 MeV	10
	>20 MeV	5
Protons, other than recoil protons, energy >2 MeV		5
Alpha particles, fission fragments, heavy nuclei		20

NOTE 1: All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

Table 2
Tissue Weighting Factors(1)

Tissue of organ	Tissue weighting factor, W_T
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05(2)(3)

NOTE 1: The values have been developed for a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

NOTE 2: For purposes of calculation the remainder is composed of the following additional tissues and organs: adrenals, brain, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer they will then be included either with a specific W_T or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

NOTE 3: In those exceptional cases in which a single one of the remainder tissues or organs receive an equivalent dose in excess of the highest dose in any of the twelve organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder.

III. BIOLOGICAL EFFECTS

A. Terminology

For the purposes of clarity, the ICRP (IC90) has introduced four terms: change, damage, harm and detriment. Changes may or may not be harmful. Damage represents some degree of deleterious change, for example to cells, but is not necessarily deleterious to the exposed individual. Harm is the term used to denote clinically observable deleterious effects that are expressed in exposed individuals (somatic effects) and in their descendants (hereditary effects). Detriment is a complex concept combining the probability, severity and time of expression of harm. In this report ICRP uses the term detriment to mean only health detriment.

So called beneficial effects of radiation, at times called "hormesis", are poorly understood and controversial. Lack of quantitative information on such effects makes it inappropriate to take them into account on the probability of stochastic effects of radiation.

The term "risk" has been used by ICRP to mean the probability of a defined deleterious effect. Now ICRP will use the term probability when that is what is being meant.

B. Deterministic Effects

Deterministic effects are deliberately excluded from this summary. No changes in thresholds for deterministic ("non-stochastic") effects were recommended for the lens of the eye, skin and extremities; deterministic limits for other tissues are given but are not being used in the calculation of annual limits on intake.

C. Stochastic Effects

Damage, including that induced by radiation to the DNA of the cell, is usually repaired by cellular processes. If the damage is not repaired correctly, the "misrepaired DNA" can give rise to a clone of cells which by a long complex process may lead to cancers or hereditary defects in individuals. To attain information on stochastic effects, epidemiological studies are customarily undertaken. However, epidemiological studies do not provide all the information needed for radiation protection.

The Japanese study is extensive (about 90,000 persons) but two-thirds of the irradiated people are still alive; estimates of forthcoming cancers must therefore be made for the surviving population. Also the doses delivered to the Japanese survivors were at high doses and high dose rates, while information is needed for occupational exposures which are often protracted and at lower dose rates. However, the Japanese study has advantages over other studies because the group studied contains both sexes and all ages and was exposed to a very wide range of doses.

In contrast, studies on medically exposed patients give rise to a different set of problems. The radiation is seldom uniform and the population is selected because of medical reasons which in turn makes it difficult to identify appropriate controls in such studies.

The simplest relationship between an increment in equivalent dose and the resulting increment in the probability of a defined stochastic effect is that of a straight line through the origin. However, epidemiological data are not sufficiently precise to confirm or exclude such a relationship. Radiobiological data often show a curvilinear dose-effect relationship (concave upwards) for low-LET radiations with the slope at low doses being less than that at high doses. At high doses the curve is complicated because the yield of effects is less due to cell killing. The results for high-LET radiation at high dose rate usually show a dose-response relationship which is concave downwards.

The direct use of epidemiological data obtained with low-LET radiation at high doses and high dose rates will probably overestimate the extrapolated risks at low doses and low dose rates. For this reason ICRP has introduced the Dose and Dose Rate Effectiveness Factor (DDREF)[1] to correct the above over-estimate. ICRP has decided to use a factor of 2 to reduce the probability of effects obtained at high doses and high dose rates to give estimates of the probability of effects at low doses and low doses rates. The DDREF has been included in probability coefficients resulting from doses below 0.2 Gy and from higher absorbed doses when the dose rate is less than 0.1 Gy per hour. The definition of low dose rates of low-LET radiation given by the ICRP is thus appreciably different from that given by UNSCEAR (see Annex C of the present ACRP report).

A second major difficulty in interpreting human data is that of estimating the number of stochastic effects yet to appear in populations being studied. In some cancers, such as leukemia and bone cancer, there is no major difficulty because the rate of appearance of new cases has dropped almost to the expected rate in the control population. For other cancers, however, the rate of appearance is still greater than that expected in the controls. To account for these differences ICRP has adopted the multiplicative risk projection model for cancers other than leukemia. In addition ICRP has used a modified multiplicative model which was used by the U.S. National Institute of Health (NIH) to develop its radioepidemiological tables. The latter takes an absolute risk from the Japanese population, transfers it to another population as an absolute risk and converts it to a relative risk in the other populations. Populations considered are those in Japan, U.S., U.K., Puerto Rico, China, and the average of all five populations.

Using the above models ICRP has estimated the probability of fatal cancer per unit effective dose (C190) and calls these estimates nominal fatality probability coefficients. In the case of the multiplicative model, the probability is corrected for other competing causes of death. Because of uncertainties in estimating cancer risks from radiation exposure and because the ICRP estimated risks for defined populations with defined exposure patterns, it calls the estimated probability of fatal cancers per unit effective dose the nominal coefficient.

Data relating to high doses and high dose rates are interpreted as suggesting a lifetime probability coefficient for a reference population of both sexes and of working age of about 8 x 10^{-2} Sv^{-1} and the corresponding value for whole populations, including children, of about 10 x 10^{-2} Sv^{-1}. Using the DDREF of 2, nominal probability coefficients are obtained as shown in Table 3. Risk coefficients for hereditary effects are also included. The new probability coefficients are compared with those given in the 1977 ICRP recommendations in Table 3.

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[1] DDREF: a factor used to derive risk estimates of low doses of low-LET radiation at low dose rates, from risk estimates calculated from data associated with large doses at high dose rates.

The current use of a multiplicative risk projection model together with a change in dosimetry at Hiroshima and Nagasaki are the major reasons that the previous risk estimates used as the ICRP basis for radiation protection (ICRP, 1977) appear now to have increased overall by about 3-4 times, since 1977.

Table 3

Nominal Risk Coefficients for Stochastic Effects (10^{-2}per Sv) (1)

Biological Effect	ICRP 1990 Whole Population	ICRP 1990 Working Population	ICRP-26
Fatal cancer	5.0	4.0	1.25
Serious Hereditary Effects:			
- in first 2 generations	---	---	0.4
- in all generations	1.0	0.6	---
Other detriment (2)	1.3	1.0	---
Total	7.3	5.6	1.65

NOTES:

(1) rounded to 2 significant figures
(2) including an adjustment for the relative loss of life expectancy due to fatal cancers and hereditary defects, and the probability and relative severity of non-fatal cancers.

In addition to providing probability estimates for fatal cancer, the ICRP attempts to quantify the detriment due to non-fatal cancer. To account for different radiosensitivities of tissues and organs on the probability of developing stochastic effects, the ICRP has given numerical values for tissue weighting factors (see Table 2). The tissue weighting factors (W_T) are based on an aggregated representation of detriment which includes four components: the probability of attributable fatal cancer, the weighted probability of non-fatal cancer, the weighted probability of severe hereditary effects, and the relative length of time lost.

The effect of including all these factors is shown in Table 4, which is taken from the ICRP 1990 draft recommendations. When non-fatal cancers and serious hereditary effects are taken into account, the nominal probability coefficients become about 7 x 10^{-2} per Sv for whole populations and 5.3 x 10^{-2} per Sv for a working population.

Table 4

Nominal Probability Coefficients for Individual Tissues and Organs
(Adapted from Table 4, IC91)

Tissue or organ	Probability of fatal cancer (10^{-2}/Sv)		Aggregated detriment: Probability of death or equivalent (10^{-2}/Sv)	
	Whole population	Workers	Whole population	Workers
Bladder	0.30	0.24	0.29	0.24
Bone marrow	0.50	0.40	1.04	0.83
Bone surface	0.05	0.04	0.07	0.06
Breast	0.20	0.16	0.36	0.29
Colon	0.85	0.68	1.03	0.82
Liver	0.15	0.12	0.16	0.13
Lung	0.85	0.68	0.80	0.64
Oesophagus	0.30	0.24	0.24	0.19
Ovary	0.10	0.08	0.15	0.12
Skin	0.02	0.02	0.04	0.03
Stomach	1.10	0.88	1.00	0.80
Thyroid	0.08	0.06	0.15	0.12
Remainder	0.50	0.40	0.59	0.47
Total	5.00	4.00	5.92	4.74
	Probability of serious hereditary disorders			
Gonads	1.00	0.6	1.33	0.80
Grand total			7.3	5.6

Estimates of the probability of fatal cancers has been extended by ICRP in 1990 to include 12 separate organs and tissues plus remainder (Table 5). Comparison of the probabilities of fatal cancers in fewer organs as given in the 1977 ICRP Publication 26 is also included in Table 5.

Table 5

Probability of fatal cancers (10^{-2} Sv^{-1}) in a population of all ages after exposure to low dose, low dose rate low-LET radiation

	ICRP 26 (1977[a])	New probabilities of fatal cancer 1990[b)]		
		International	U.S.A.:	
U.S.A.:		average	multiplicative model	NIH model
Bone marrow	.20	.50	.54	.40
Bone surface	.05	.05	(.05)	(.05)
Lung	.20	.85	1.15	.53
Thyroid	.05	.08	(0.08)	(0.08)
Breast	.25	.20	.42	.15
Colon		.85	1.79	.82
Oesophagus		.30	.08	.11
Stomach		1.10	.18	1.38
Liver		.15	(.15)	(.15)
Bladder		.30	.43	.21
Ovary		.10	.17	.10
Skin		.02	(.02)	(.02)
Remainder	.50	.50	.54	.35
Total	1.25	5.0	5.6	4.35

a IC77 ICRP Publication 26. Recommendations of the International Commission on Radiological Protection. Annals of the ICRP, Vol. 2, No. 3 (1977).

b IC91 Recommendations of the International Commission on Radiological Protection. International Commission on Radiological Protection (1991).

The last 2 columns in Table 5 for the U.S. population were derived from Tables 14A and 14B of Appendix B in the Nov. 1990 ICRP draft, applying a DREF of 2. The bracketed values for 4 tissues (bone surface, thyroid, liver, and skin) are not derived from the data on Japanese bomb survivors but were inserted for ease of comparison with the recommended international averages given in Table 5. This table provides some indication of the uncertainties in the probability coefficients for individual organs and tissues, depending on the model used to derive these coefficients. The 8-fold variation in the case of stomach cancer probably reflects the fact that stomach cancer is normally more prevalent in Japan than in the U.S.A. The ICRP draft commented "unfortunately, there is no general agreement on which, if any, transfer method is to be preferred or indeed whether the same method should apply to each cancer site. Nor is there any specific reference population with which ICRP should deal." The ICRP draft chose to average all the results obtained with the multiplicative and NIH risk projection and transfer models, as applied to the populations of Japan, U.S.A., U.K., Puerto Rico, and China in order to obtain the recommended international averages shown in Table 5.

Other models would of course yield somewhat different results. The predicted data for the U.S. population using a simple relative risk projection model are appreciably lower in the case of stomach and oesophageal cancer and appreciably higher in the case of breast and colon cancer than would be indicated by the ICRP weighting factors.

IV. DOSE LIMITS

The ICRP draft modified the dose limits and now recommends the following dose limits as given in Table 6.

"The ICRP has reached the judgement that its dose limit should be set in such a way and at such a level that the total effective dose received in a full working life would be prevented from exceeding about 1 Sv received moderately, uniformly year by year and that the application of its system of radiological protection should be such that this figure would only rarely be approached".

To assess individual risk the probability of incurring the dose times the lifetime probability of attributable cancer death from that dose have to be considered and yield a single probability when multiplied together. Thus a restriction corresponding to the dose limit can be expressed in the form of a risk limit, that is a limit on the fatality coefficient. The ICRP does not recommend an annual risk limit for individuals.

Table 7 shows the calculated loss of life expectancy due to continued occupational exposures at the recommended limit every year for 47 years starting at age 18. The average loss of life expectancy due to potential fatal cancers induced by 20 mSv per year as calculated in 1990 is similar to the corresponding values calculated for 50 mSv per year in 1977. The probability of death due to radiation-induced cancers was calculated in the 1990 ICRP draft to be maximal at age 78 on the basis of the multiplicative risk projection model as compared to age 68 using the additive risk projection model. The loss of life expectancy per radiation-induced fatal cancer was calculated by the ICRP to be about 13 years for the multiplicative risk projection model used in 1990, as compared to about 20 years for the 1977 data using the absolute risk model. The decrease in annual limit from 50 to 20 mSv per year is thus sufficient to offset the recent increase in estimated probability of radiation-induced cancer when expressed in terms of attributable loss of life expectancy.

The aggregated detriment coefficients are, as noted above, somewhat greater than the probability of radiation-induced fatal cancers alone. However, the probability of radiation-induced fatal cancers accounts for about 70% of the total aggregated detriment coefficients recommended by ICRP both in 1977 (IC77a) and in the 1990 draft (IC90). It might also be noted that the average loss of life expectancy of 0.5 years due to potential fatal cancers induced by an occupational exposure of 20 mSv per year (Table 6) is equivalent to the average loss of life expectancy attributable to a fatal accident rate in industry of 3 per 10^4 workers per year, assuming an average loss of 35 years of life expectancy per fatal accident. This topic has been considered in more detail in previous ICRP publications on index of harm (see ICRP Publications 27 and 45).

Table 6

Recommended Dose Limits (1)
(Adapted from Table 6 of IC91)

Application	Dose Limit Occupational	Public
Effective dose	20 mSv per year averaged over 5 consecutive years with with a maximum of 50 mSv in any 1 year	1 mSv per year, averaged over any 5 consecutive years
Annual equivalent dose		
in the lens of the eye	150 mSv	15 mSv
the skin (2)	500 mSv	50 mSv
the hands and feet	500 mSv	---
Mean dose to the surface of mother's abdomen	2 mSv after diagnosis of pregnancy (3)	

NOTE 1: The limits apply to the sum of the doses from external exposure in the specified period and the 50-year committed dose (70-year for public exposure) from intakes in the same period.

NOTE 2: The annual limit of 500 mSv is averaged over areas of no more than 1 cm^2, regardless of the area exposed.

NOTE 3: During the remainder of the pregnancy the intake of radionuclides is limited to about 1/20 of the annual limit on intake.

Table 7

Loss of life expectancy due to fatal cancers induced by 47 years of occupational exposures at the recommended limits

	1977 data	1990 estimates
Annual dose (mSv)	50	20
Lifetime occupational dose (Sv)	2.35	0.94
Probability of death due to fatal cancers induced by occupational exposures (%) (a)	2.9	3.8
Time lost per radiation-induced fatal cancer (years) (b)	20	13
Average loss of life expectancy per worker (years)	0.6	0.5

(a) Probability of fatal cancer per Sv taken as 1.25×10^{-2} in 1977 and as 4×10^{-2} in 1990.

(b) Years lost per cancer taken from paragraphs 83 and 156 of the 1990 ICRP recommendations. The value of 20 years loss of life expectancy based on an absolute risk model is consistent with the data given in Annex B but not with the data in Table 5 of the 1990 ICRP recommendations. This difference is discussed in paragraph C69 of Annex C of the 1990 ICRP recommendations (IC91).

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III. GLOSSARY

absolute risk: an expression of excess risk based on the assumption that the excess risk from radiation exposure adds to the underlying (baseline) risk by an increment dependent on dose but independent of the underlying natural risk

absorbed dose: the mean energy imparted by ionizing radiation to an irradiated medium per unit mass. Units: gray (Gy)

ACRP: Advisory Committee on Radiological Protection, Atomic Energy Control Board of Canada

activity: the number of nuclear transformations of a radioactive nuclide per unit time; units: becquerel (Bq)

adenosarcoma: a mixed tumor which consists of a substance like embryonic connective tissue together with glandular elements

AECB: Atomic Energy Control Board of Canada

ALARA: all exposures shall be kept As Low As Reasonably Achievable, economic and social factors being taken into account

ankylosing spondylitis: arthritis of the spine

annual limit on intake (ALI): the amount of radioactive substance taken into the body in one year that will result in a "committed dose" equal to either the stochastic or non-stochastic dose limit; different ALI values apply to "intake" by inhalation and ingestion, and different values apply to atomic radiation workers and members of the public

autosome: any ordinary paired chromosome as distinguished from a sex chromosome

background radiation: the amount of radiation to which a member of the population is exposed from natural sources, such as terrestrial radiation due to naturally occurring radionuclides in the soil, cosmic radiation originating in outer space, and naturally occurring radionuclides deposited in the human body

baseline rate: the cancer experience observed in a population in the absence of the specific agent being studied; the baseline rate might, however, include cancers from a number of other causes, such as smoking, background radiation, etc.

becquerel (Bq): a unit of activity equal to one radioactive disintegration per second

BEIR: Biological Effects of Ionizing Radiation; an advisory committee which is funded by the U.S. Environmental Protection Agency and which reports to the U.S. National Academy of Sciences

blastomere: one of the cells produced by cleavage of a fertilized ovum, forming the blastoderm

building shielding factor: a correction factor that takes into account the shielding provided from being inside a building, or outside but close to a building that can attenuate some of the incident radiation

cancer: a malignant tumor of potentially unlimited growth, capable of invading surrounding tissue or spreading to other parts of the body by metastasis; some cancers are fatal while others in certain tissues such as skin and thyroid can usually be cured by medical intervention

carcinogen: an agent that may cause cancer; ionizing radiations are physical carcinogens; there are also chemical and biologic carcinogens

carcinoma: a malignant tumor (cancer) of epithelial origin

case-control study: an epidemiological study in which people with disease and a similarly composed group of people without disease are compared in terms of exposures to a putative causative agent

cerebellum: a part of the brain concerned with the coordination of movements

chromosome: one of several rod-shaped bodies in the nucleus of a cell which contain the genes or hereditary factors

cleavage: the mitotic segmentation of the fertilized ovum, the size of the zygote remaining unchanged and the cleavage cells, or blastomeres, becoming smaller and smaller with each division

cohort study: or follow-up study; an epidemiological study in which groups of people are identified with respect to the presence or absence of exposure to a disease-causing agent and the outcomes in terms of disease rates are compared

collective dose: the sum of the individual radiation doses received by a group of person, expressed in person-sieverts

committed dose: the total dose received from a radioactive substance in the body during 50 years following the intake of this substance in the case of workers and 70 years following the intake in the case of members of the population

confidence interval: a measure of the reliability of a risk estimate; a 90% confidence interval means that 9 times out of 10 the estimated risk would be within the specified interval

congenital: existing at, and usually before birth; referring to conditions present at birth, regardless of their causation

cortex: the convoluted layer of gray substance that covers each cerebral hemisphere

DDREF: a factor used to derive risk estimates of low doses of low-LET radiation at low dose rates, from risk estimates calculated from data associated with large doses at high dose rates

deterministic: see non-stochastic

detriment: the mathematical expectation of individual or collective harm incurred from exposure to radiation, taking into account the probability of each type of deleterious health effect and the severity of each effect

diploid: having two sets of chromosomes, as normally found in the somatic cells of species such as man

dose: a dose of radiation, either an "absorbed dose" or "dose equivalent", depending on the context

dose commitment: the sum of all doses received following the release or intake of a given amount of a radioactive material, summed over a period of time extending to infinity

dose effect (dose-response) model: a mathematical formulation of the way the effect (or biological response) depends on dose

dose equivalent: the "absorbed dose" multiplied by a "quality factor" or radiation weighting factor to account for the different potential for injury of different types of radiation, where the "absorbed dose" is the amount of energy absorbed the body, or in an organ or tissue of the body, due to exposure to ionizing radiation, divided by the respective mass of the body, organ or tissue; a gray of "absorbed dose" multiplied by the appropriate "quality factor" or radiation weighting factor yields the "dose equivalent" in units of "sieverts"

dose limit: a legal limit on radiation dose

dose rate: the quantity of dose per unit time

doubling dose: the amount of radiation needed to double the natural incidence of a genetic or somatic anomaly

DREF: a factor used to derive risk estimates of exposure to low-LET radiation at low dose rates, from risk estimates calculated from data associated with exposures at high dose rates

effective dose: see effective dose equivalent

effective dose equivalent: the sum of the dose equivalents received by different tissues of the human body, each multiplied by the internationally recommended values of the "tissue weighting factors" assigned to each respective tissue; the weighting factor reflects the probability that a unit dose of radiation to the tissue in question will result in a cancer in the exposed person or a serious genetic disorder in the descendants of the exposed person; the sum of the weighting factors for all tissues of the body is 1.0

embryo: the developing organism from one week after conception to the end of the second month

epidemiology: the study of the determinants of the frequency of disease in man; the two main types of epidemiological studies of chronic disease are cohort (or follow-up) studies and case-control (or retrospective) studies

equivalent dose: see dose equivalent

exposure: exposure to an internal or external source of radiation, or, depending on the context, the product of the concentration of a radioactive substance in air (or the dose rate from a source of radiation) and the duration of exposure to that concentration (or dose rate)

fertilization: union of male sperm and female ovum leading to the development of a new individual

fluence: the number of particles passing through a sphere of given cross-sectional area, per unit area

fluoroscopy: a method of visualizing internal structures by directing X-rays through an object (e.g., part of the body) onto a fluorescent screen

fractionation: the delivery of a given total dose of radiation as several smaller doses, separated by intervals of time

free-in-air kerma: the "kerma" in tissue in air over bare ground, i.e. no person present and not in or near a building

foetus: the developing youth in the human uterus after the end of the second month

gamete: one of two cells, male and female, whose union is necessary, in sexual reproduction, to initiate the development of a new individual

gastrulation: the process by which the young embryo acquires its three germ layers

gene: the biologic unit of heredity, self-reproducing and located in a definite position (locus) on a particular chromosome

genetic disorder or disease: an inherited disease; alternatively, a serious individual deviation from the average characteristics of human populations, which requires medical attention at some time in the person's life and which is genetic or partially-genetic in origin

gestation: the period of development of the young in the uterus from the time of fertilization of the ovum

gonad: one of the primary sex glands which include the ovaries in a woman and the testes in a man

gonosomal: referring to the sex chromosomes

gray (Gy): the special name of the unit of "absorbed dose"; (1 Gy = 1 joule/kg)

haploid: having a single set of chromosomes, as normally carried by a gamete

high-LET radiation: radiation characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus

IAEA: International Atomic Energy Agency

ICRP: International Commission on Radiological Protection

incidence: or incidence rate; the rate of occurrence of a disease within a specified period of time, often expressed as number of cases per 100,000 individuals per year

inheritable injury: hereditary defects that are genetically transmitted from a parent to a child and which may appear amongst the children and subsequent generations of a parent whose gonads (ovaries or testes) have been exposed to radiation anytime before the conception of children

in utero: in the womb; i.e., before birth

in vitro: in culture or in the test-tube (as opposed to in vivo, in the living individual)

in vivo: in the living organism

ionizing radiation: radiation which is sufficiently energetic to dislodge electrons from an atom and thus to produce ions in an otherwise non-ionized gaseous medium

joule (J): the name of the unit of energy in the "S.I." system of units

kerma: the sum of the initial kinetic energies of all those primary charged particles which are generated per unit mass of material by the incident radiation

latent period: the period of time between exposure and expression of the disease. After exposure to a dose of radiation, there is a delay in several years (the minimum latent period) before any radiation-induced cancers are seen

LET: linear energy transfer, usually expressed as units of energy deposited per unit length of the track of an ionizing ray or particle

life-span study (LSS): life-span study of registered Japanese atomic bomb survivors

life table: a table showing the number of persons who, of a given number born or living at a specified age, live to attain successive higher ages, together with the numbers who die in each age interval

linear energy transfer (LET): average amount of energy lost per unit track length

low-LET radiation: radiations such as X or gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus

linear model: also, linear dose-effect relationship; expresses the effect (e.g., mutation or cancer) as a direct (linear) function of dose

linear-quadratic model: or linear-quadratic dose-effect relationship; expresses the effect (e.g., mutation or cancer) as partly directly proportional to the dose (linear term) and partly proportional to the square of the dose (quadratic term); the linear term will predominate at lower doses, the quadratic term at higher doses

lymphoma: a general term applied to any neoplastic disorder of the lymphoid tissue

MIRD phantom: a mathematical representation of the human body in which specific organs and tissues are represented by simplified volumes such as spheres and cylinders; doses to body tissues can be calculated for known incident radiations by combining the MIRD phantom with radiation transport programs

mitosis: a method of division of a cell, consisting of a complex of various processes, by means of which the two daughter nuclei normally receive identical complements of the number of chromosomes characteristic of the somatic cells of the species

morphogenesis: the evolution and development of form, as the development of the shape of a particular organ or part of the body

mortality (rate): the rate to which people die from a disease, e.g., a specific type of cancer, often expressed as number of deaths per 100,000 per year

NCRP: National Council on Radiation Protection and Measurements, USA

neoplasms: any new and abnormal growth, such as a tumor; neoplastic disease refers to any disease that forms tumors, whether malignant or benign

non-stochastic effects: effects whose severity is frequently a function of dose and for which doses above some threshold dose is required

NRPB: National Radiological Protection Board, UK

nuclear facility: a nuclear reactor, a sub-critical nuclear reactor, a particle accelerator, a uranium or thorium mine or mill, a plant for the separation, processing, re-processing or fabrication or fissionable substances, a plant for the production of deuterium or deuterium compounds, and a facility for the disposal of prescribed substances, including all land, buildings and equipment that are connected or associated with such reactor, accelerator, plant or facility

neuroglia: support cells associated with neurons

neuron: a nerve cell with its processes, collaterals and terminations regarded as a structural unit of the nervous system

neutron: an uncharged subatomic particle capable of producing ionizing radiations as an indirect consequence of its collisions with atomic nuclei

occupational accidents: fatal or non-fatal injuries to workers which occur at work

occupational dose or occupational exposure: a radiation dose or an exposure to radiation received while employed as a radiation worker

organ: a structure of the body such as the lungs, gonads or thyroid gland that performs some specialized function

organogenesis: the origin and development of body organs

organ shielding factor: a correction factor that takes into account the shielding provided by the body that attenuates the transmission of some of the incident radiation and consequently alters the actual doses received by different internal organs of the body

ovum: the female reproductive cell which, after fertilization, develops into a zygote

perinatal: pertaining to or occurring in the period shortly before or after birth

person-sievert (p-Sv): the unit of population exposure obtained by summing individual dose equivalent values for all people in the exposed population; the unit of "collective dose"

person-years-at-risk (PYAR): the number of persons exposed multiplied by the number of years after exposure

pia mater: the innermost of the three membranes (meninges) covering the brain and spinal cord

prevalence: the number of cases of a disease in existence at a given time per unit population, usually 100,000 persons

projection model: a mathematical model that simultaneously described the excess cancer risk at different levels of some factor such as dose, time after exposure, or baseline level of risk, in terms of a parametric function of that factor; it becomes a projection model when data in a particular range of observations is used to assign values to the parameters in order to estimate (or project) excess risk for factor values outside that range

promoter: an agent which is not by itself carcinogenic, but which can amplify the effect of a true carcinogen by increasing the probability of late-stage cellular changes needed to complete the carcinogenic process

quadratic-dose model: a model which assumes that the excess risk is proportional to the square of the dose

quality factor (Q): an LET dependent factor by which absorbed doses are multi-plied to obtain (for radiation protection purposes) a quantity which corresponds more closely to the degree of biological effect produced by X- or low-energy gamma rays; dose in Gy x Q = dose equivalent in Sv

radiation weighting factor: see quality factor

radiogenic: caused by radiation

radiosensitivity: relative susceptibility of cells, tissues, organs, and organisms to the injurious action of radiation; radiosensitivity and its antonym, radioresistance, are used in a comparative sense rather than an absolute one

relative biological effectiveness (RBE): biological potency of one radiation as compared with another to produce the same biological endpoint; it is numerically equal to the inverse of the ratio of absorbed doses of the two radiations required to produce equal biological effect; the reference radiation is often 200 kV X-rays.

relative risk: an expression of excess risk relative to the underlying (baseline) risk; if the excess equals the baseline risk the relative risk is 2

RERF: Radiation Effects Research Foundation; a bi-nationally funded Japanese foundation chartered by the Japanese Welfare Ministry under an agreement between the U.S.A. and Japan; the RERF is the successor to the ABCC (Atomic Bomb Casualty Commission)

risk coefficient: the increase in the annual incidence or mortality rate per unit dose: (1) absolute risk coefficient is the observed minus the expected number of cases per person year at risk for a unit dose; (2) the relative-risk coefficient is the fractional increase in the baseline incidence or mortality rate for a unit dose

risk estimate: the number of cases (or deaths) that are projected to occur in a specified exposed population per unit dose for a specified exposure regime and expression period; number of cases per person-gray or, for radon, the number of cases per person cumulative working-level month

sarcoma: a malignant growth arising in tissue of mesodermal origin (connective tissue, bone, cartilage or striated muscle)

sex-linked mutation (or X-linked): a mutation associated with the X chromosome. It will usually only manifest its effect in males (who have only a single X chromosome).

scalar fluence: see "fluence"

sievert (Sv): the special name of the unit of "dose equivalent" and of "effective dose equivalent"

spermatozoon: a mature male germ, the specific output of the testes

standard mortality ratio (SMR): standard mortality ratio is the ratio of the disease or accident mortality rate in a certain specific population compared with that in a standard population; if the ratio is based on 100 percent for the reference standard, an SMR of 200 means that the test population has twice the mortality from that particular cause of death

stochastic effects: biological effects that may occur at random amongst an exposed group of people, with the probability of occurrence being proportional to the effective dose equivalent, usually without a threshold; cancer and inheritable injury are the stochastic effects of primary concern

synapse: the region of contact between processes of two adjacent neurons, forming the place where a nervous impulse is transmitted from one neuron to another

teratogen: an agent or factor that causes the production of physical defects in the developing embryo

thalamus: a part of the brain which is the main relay centre for sensory impulses to the cerebral cortex

threshold dose: the minimum dose below which there is no given biological response or effect

tissue: group of similar cells which together perform certain specialized functions (e.g,, bond marrow, bone surfaces, and eye lenses)

tissue weighting factor: a factor by which the dose equivalent received by any organ or tissue of the body is multiplied in order to account for the risk of fatal cancer or inheritable injury resulting from irradiation of that organ or tissue compared to the total risk of such stochastic effects resulting from the receipt of an equal dose equivalent by the whole body

UNSCEAR: United Nations Scientific Committee on the Effects of Atomic Radiation

WHO: World Health Organization

WLM: Working Level Month, originally defined as the exposure received while working for 170 hours in an atmosphere containing 100 picocuries (3.7 becquerel) of radon-222 per litre in equilibrium with its short-lived daughters

X-ray: an electromagnetic radiation emitted during certain rearrangements of the electron shells of an atom; X-rays may be produced during radioactive decay or from the slowing down of fast electrons in any material; they are usually obtained from X-ray machines in which a metallic target is bombarded with fast electrons in a vacuum

zygote: the cell resulting from the fusion of two gametes; the fertilized ovum

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